ECG DIAGNOS

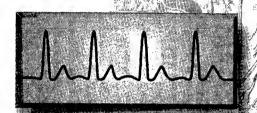
A Self-Assessment Workbook

Edward K. Chung, M.

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Blackwell Wissenschafts-Verlag GmbH, Kurfürstendamm 57, I0707 Berlin, Germany

Blackwell Science KK, MG Kodenmacho Building, 7-10 Kodenmacho Nihombashi, Chuo-ku, Tokyo 104, Japan

Distributors:

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Acquisitions: Chris Davis

Cover Design: Meral Dabcovich, VisPer

Production: Erin Whitehead

Typeset by Best-set Typesetter Ltd., Hong Kong

Manufacturing: Lisa Flanagan

Printed and bound by Sheridan Books, Inc.

Printed in the United States of America

00 01 02 5 4 3 2 1

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Library of Congress Cataloging-in-Publication Data

Chung, Edward K.

ECG diagnosis: a self-assessment workbook/by Edward K. Chung.

p.; cm.

Companion v. to: Pocket guide to ECG diagnosis. c1996.

Companion v. to: Pocket guide to ECG diagnosis and self assessment CD-ROM

[computer file]. c1997.

ISBN 0-86542-587-6

1. Electrocardiography—Handbooks, manuals, etc. 2. Heart—Diseases—Diagnosis—Handbooks, manuals, etc. 3. Electrocardiography—Examinations, questions, etc. 4. Heart—Diseases—Diagnosis—Examinations, questions, etc. I. Title.

[DNLM: 1. Electrocardiography—Examination Questions. WG 18.2 C559e 2000]

RC683.5.E5 C4636 1996 Suppl.

616.1'207547-dc21

99-049376

to
My Wife, Lisa
Children, Linda and Christopher
Son-in-law, James
Daughter-in-law, Sue
and

Grandchildren, Nicholas and Jacqueline

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Preface

ECG Diagnosis: A Self-Assessment Workbook provides an educational self-assessment guideline for the interpretation of various electrocardiographic abnormalities. In this book, 250 common and clinically pertinent ECG abnormalities are included. Thus, any ECG tracings showing extremely unusual and rare ECG abnormalities or complex cardiac arrhythmias have been purposely omitted.

ECG Diagnosis: A Self-Assessment Workbook will be extremely valuable to medical students, medical residents, cardiology fellows, and all primary care physicians (e.g., internists, family medicine physicians, and emergency room physicians). In addition, this book will be useful to cardiac care nurses, anesthesiologists, rehabilitation medicine physicians, and paramedical personnel.

You may work your way through the exercises in this book consecutively for a comprehensive assessment of your diagnostic skills, at random for a more "real life" challenge, or, to test your knowledge in a particular area (*Atrial Arrhythmias*, for example), simply turn to that section. For each case, carefully review the ECG image and then record your interpretation in the space provided. You may find the *Key to the Diagnostic Criteria* and *Abbreviations Key* provided at the front of the book to be helpful. To check your interpretation, turn to the corresponding case in the *Case Descriptions* section at the back of the workbook.

ECG Diagnosis: A Self-Assessment Workbook will be a companion volume to Pocket Guide to ECG Diagnosis and ECG Diagnosis and Self-Assessment CD-ROM by the same author.

I would like to express my sincere gratitude to my daughter-in-law, Sue, for her valuable editorial work to complete this book. In addition, the endless cooperation of the editorial staff of Blackwell Science is deeply appreciated.

Edward K. Chung, MD Bryn Mawr, PA

ABBREVIATIONS KEY

AF: Atrial fibrillation

APC: Atrial premature contraction

ASD: Atrial septal defect AT: Atrial tachycardia

AVC: Aberrant ventricular conduction

A-V: Atrioventricular

A-V JEB: A-V junctional escape beat A-V JER: A-V junctional escape rhythm

A-V JPC: A-V junctional premature

contraction

A-V JT: A-V junctional tachycardia

BBBB: Bilateral bundle branch block

BFB: Bifascicular block

BTS: Brady-tachyarrhythmia syndrome

BVH: Biventricular hypertrophy CAD: Coronary artery disease

CCU: Coronary care unit

CHF: Congestive heart failure

COPD: Chronic obstructive pulmonary disease

CPR: Cardiopulmonary resuscitation

CSS: Carotid sinus stimulation

DC shock: Direct current shock

DI: Digitalis intoxication

DPLMI: Diaphragmatic posterolateral myocardial infarction

ECG: Electrocardiogram

ERP: Early repolarization pattern

HLVV: High left ventricular voltage

IHSS: Idiopathic hypertrophic subaortic stenosis

JEB: Junctional escape beat

JER: Junctional escape rhythm

JPC: Junctional premature contraction

JT: Junctional tachycardia

JTWP: Juvenile T wave pattern

LAD: Left axis deviation

LAE: Left atrial enlargement

LAHB: Left anterior hemiblock

LBBB: Left bundle branch block

LGL syndrome: Lown-Ganong-Levine

syndrome

LPHB: Left posterior hemiblock

LV: Low voltage

LVH: Left ventricular hypertrophy MAT: Multifocal atrial tachycardia

MI: Myocardial infarction

MS: Mitral stenosis

MVPS: Mitral valve prolapse syndrome

NSR: Normal sinus rhythm

PAT: Paroxysmal atrial tachycardia

PDA: Patent ductus arteriosus

PE: Pulmonary embolism

PS: Pulmonic stenosis

RAD: Right axis deviation

RAE: Right atrial enlargement

RB: Reciprocal beat

RBBB: Right bundle branch block

RHD: Rheumatic heart disease

RVH: Right ventricular hypertrophy

S-A: Sinoatrial

SSS: Sick sinus syndrome

SVT: Supraventricular tachycardia

TFB: Trifascicular block

V-A: Ventriculoatrial

VEB: Ventricular escape beat

VER: Ventricular escape rhythm

VF: Ventricular fibrillation

VPC: Ventricular premature contraction

VPSA: Ventriculophasic sinus arrhythmia

VSD: Ventricular septal defect

VT: Ventricular tachycardia

WAP: Wandering atrial pacemaker

WPW syndrome: Wolff-Parkinson-White

syndrome

KEY TO THE DIAGNOSTIC CRITERIA

Aberrant ventricular conduction: Case 107

Atrial dissociation: Case 215

Atrial premature contraction (APC): Case 106

Ashman's phenomenon: Case 107 A-V block classification: Case 173 A-V dissociation causes: Case 139

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Case 23

Biventricular hypertrophy (BVH): Case 17

Causes of A-V dissociation: Case 139

Causes of broad QRS complex: Case 158

Causes of ventricular pause: Case 110

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Electrical alternans: Case 249

Fascicular rhythm and tachycardia: Case 214

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Hypothermia: Case 51

Junctional premature contraction (JPC):

Case 130

Left anterior hemiblock (LAHB): Case 36

Left atrial enlargement (LAE): Case 6

Left atrial rhythm: Case 217

Left bundle branch block (LBBB): Case 26 Left posterior hemiblock (LPHB): Case 37 Left ventricular hypertrophy (LVH): Case 11

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Malfunctions of artificial pacing: Case 226 Mobitz type I (Wenckebach) A-V block: Case 174

Mobitz type II A-V block: Case 183

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Myocardial infarction (M1): Case 65

Non-Q wave M1: Case 82 Normal sinus rhythm: Case 1 Normal variants: Case 3

Parasystole: Case 208

Pericarditis: Case 242 Pseudo MI: Case 84

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Right bundle branch block (RBBB):

Case 19

Right ventricular hypertrophy (RVH):

Case 6

Right ventricular M1: Case 81

S-A block (type 1): Case 100

S-A block (type II): Case 99

Sick sinus syndrome (SSS): Case 96

Subendocardial injury: Case 63

Subendocardial ischemia: Case 54

Subepicardial ischemia: Case 55

Subepicardial injury: Case 59

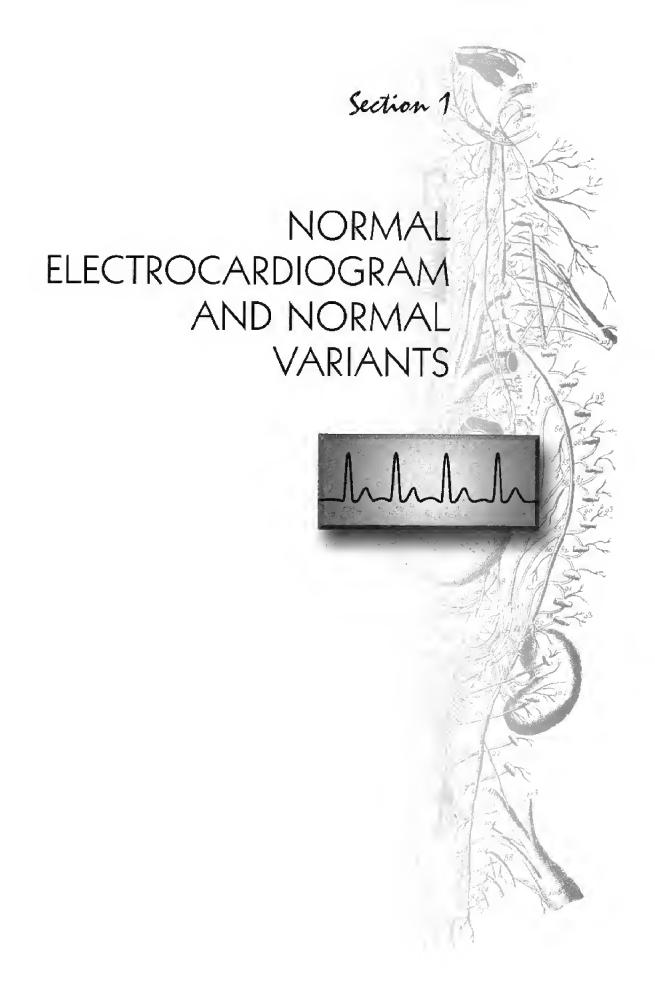
Ventricular parasystole: Case 208

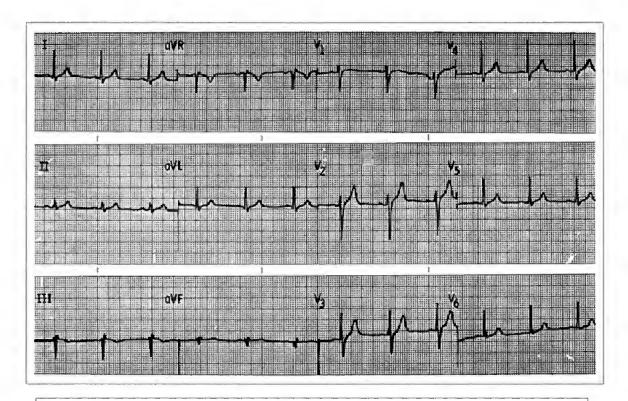
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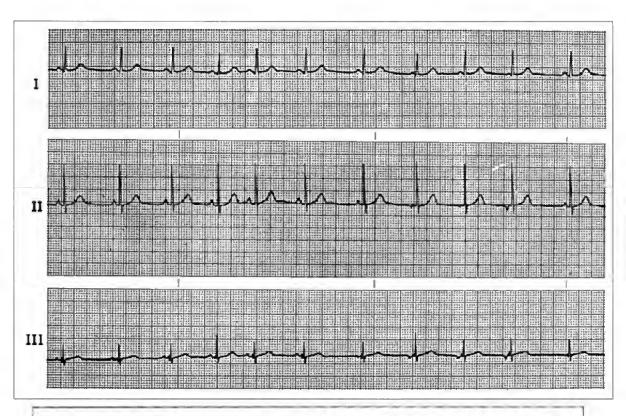
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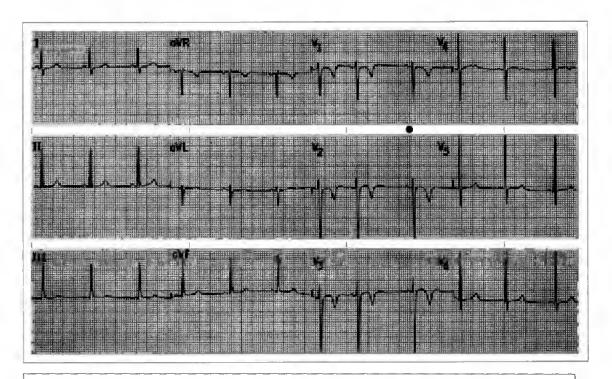




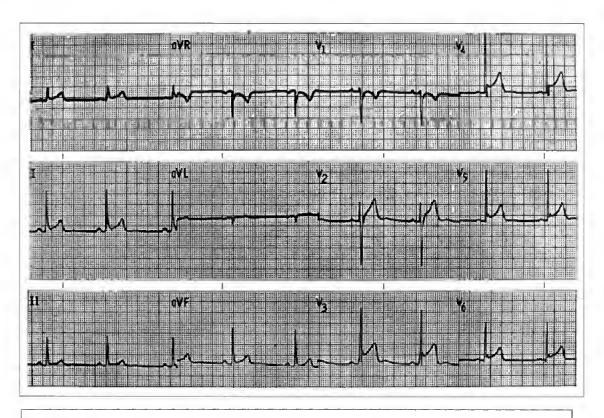




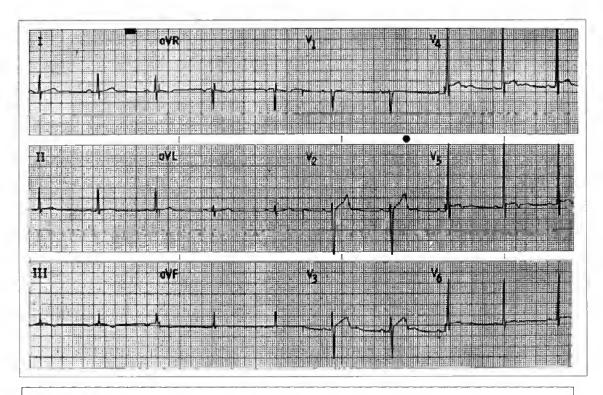




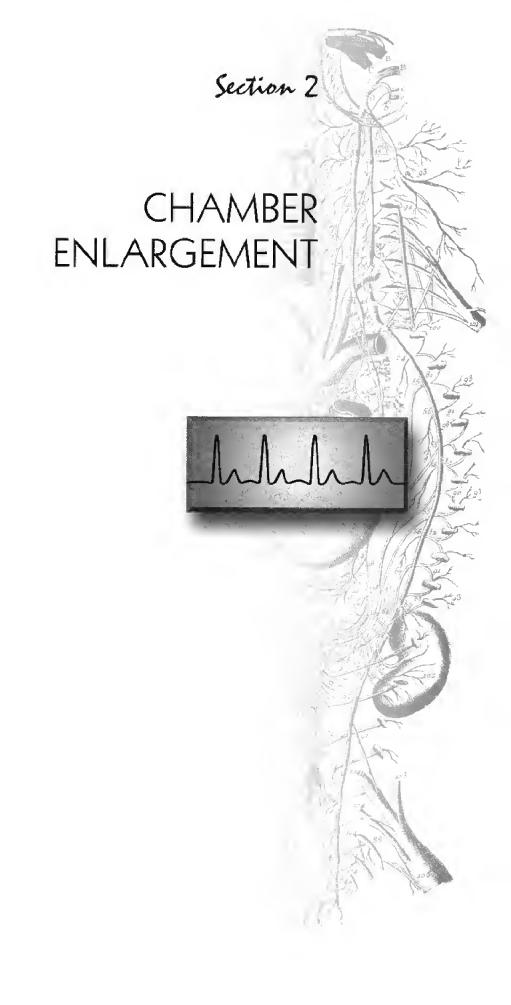


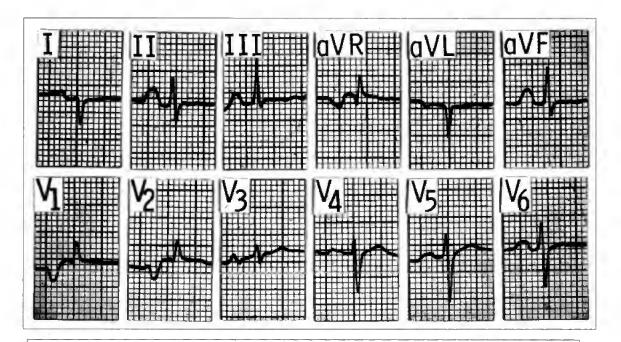




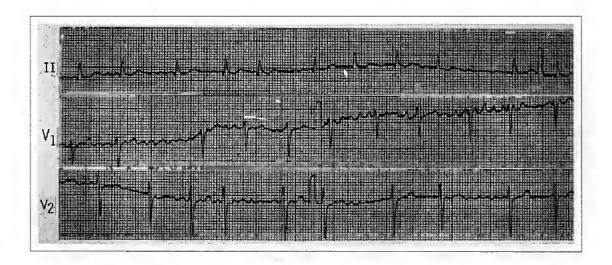




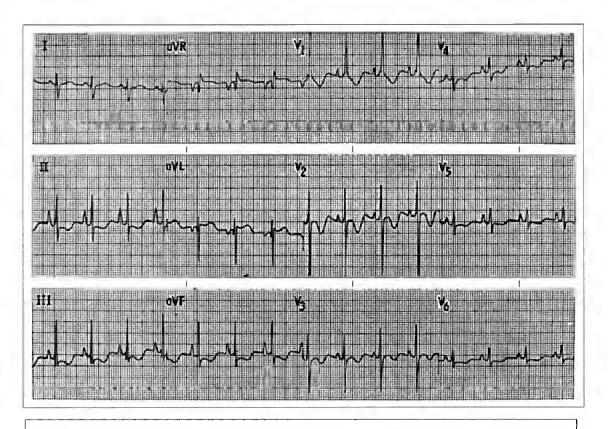




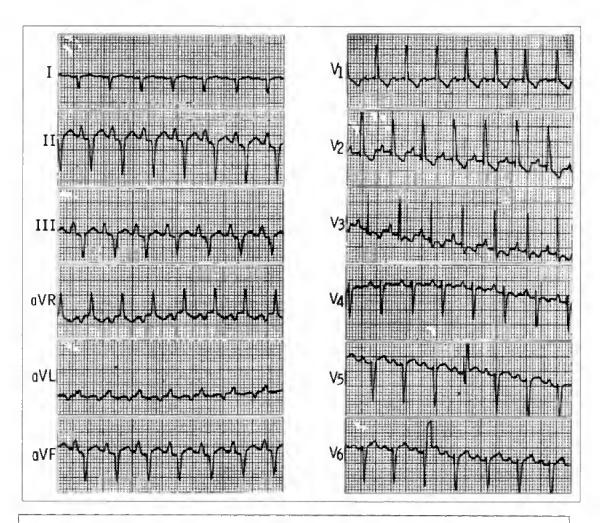




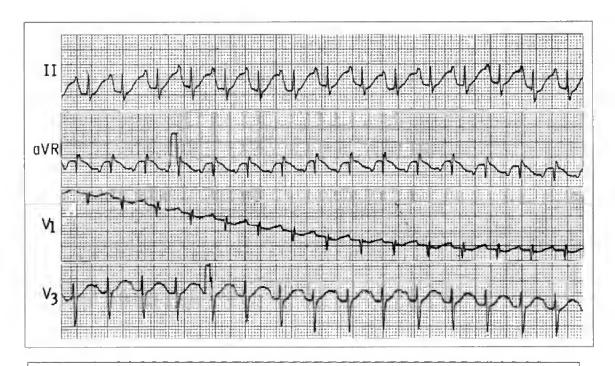




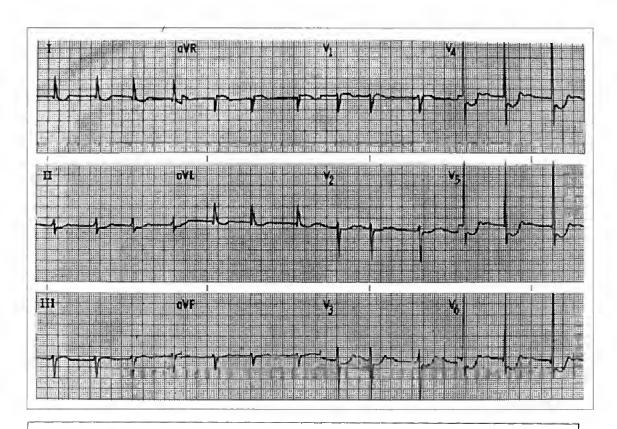




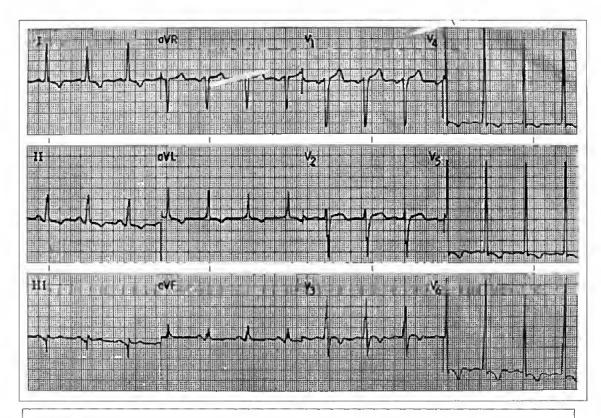




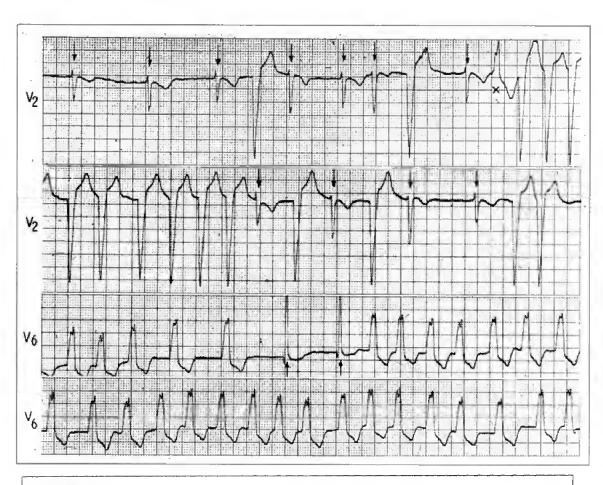






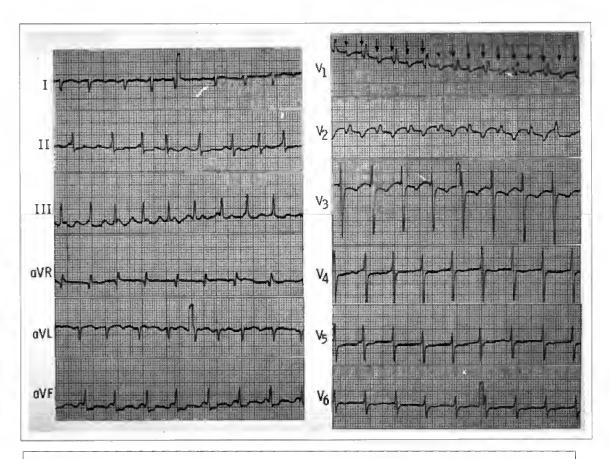




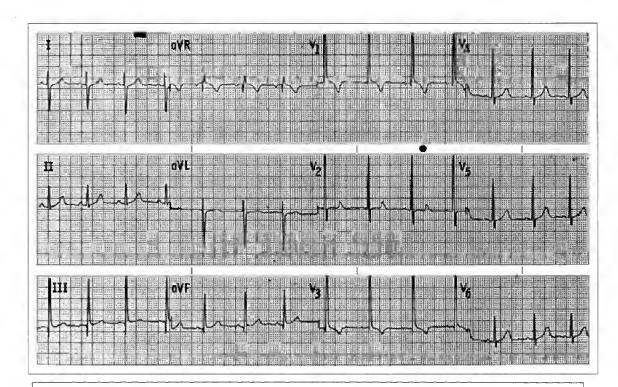




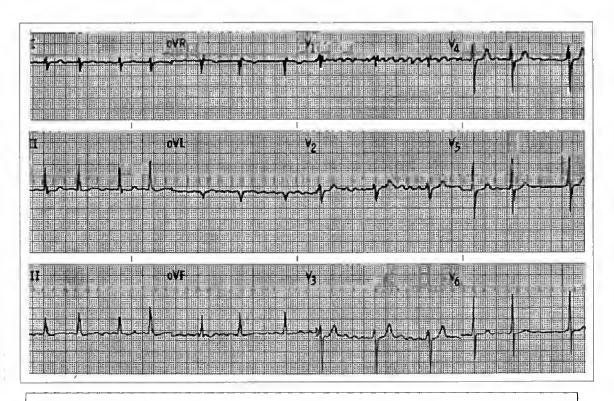




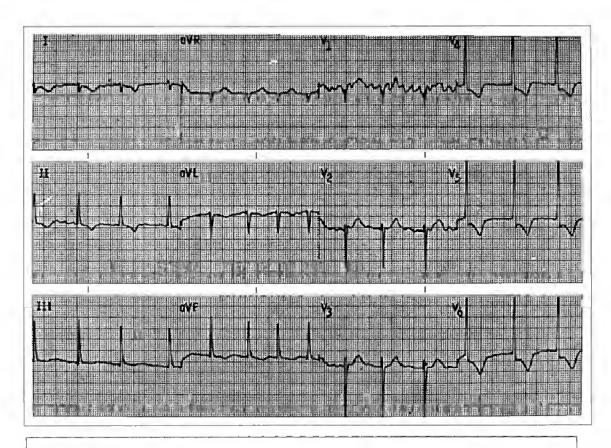




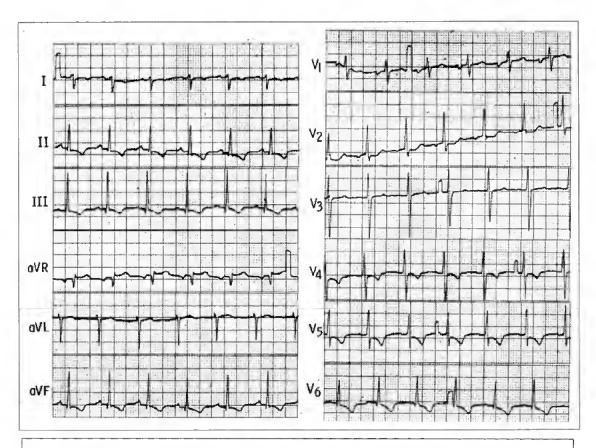




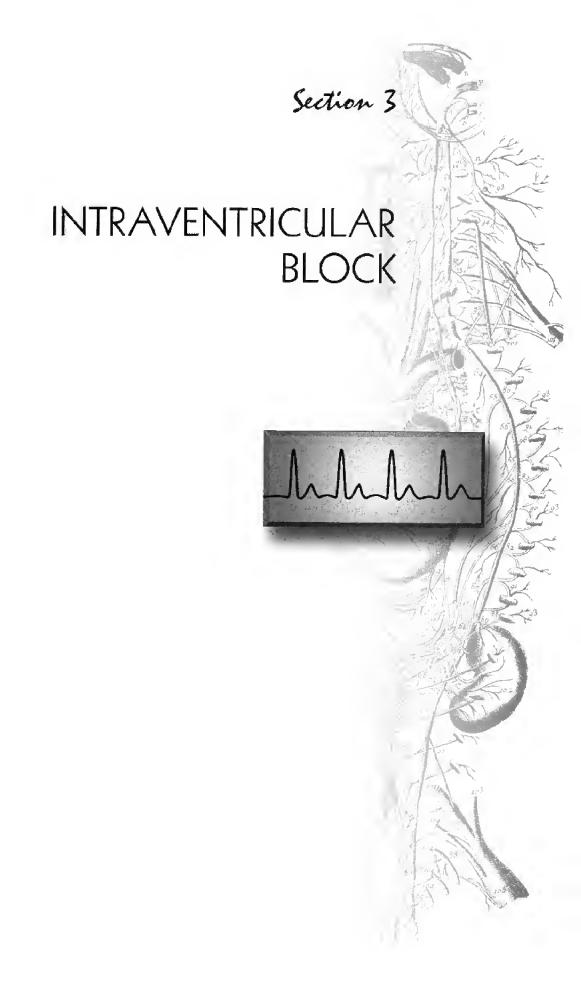


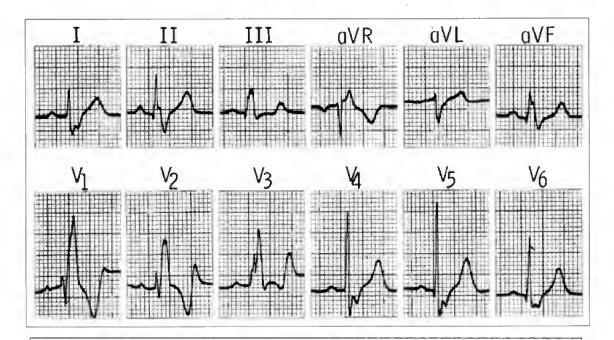




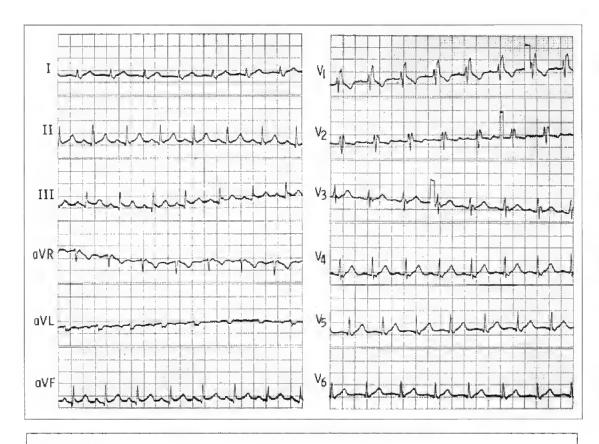




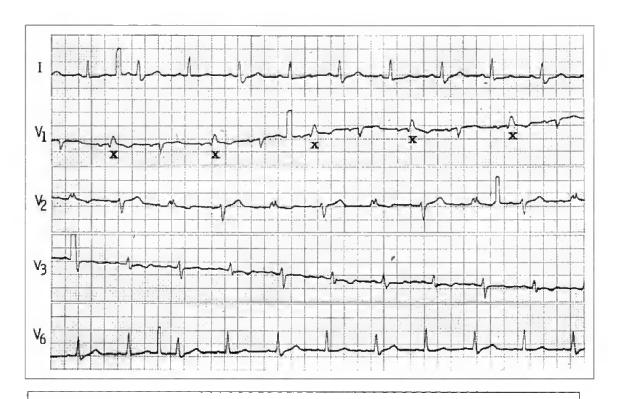


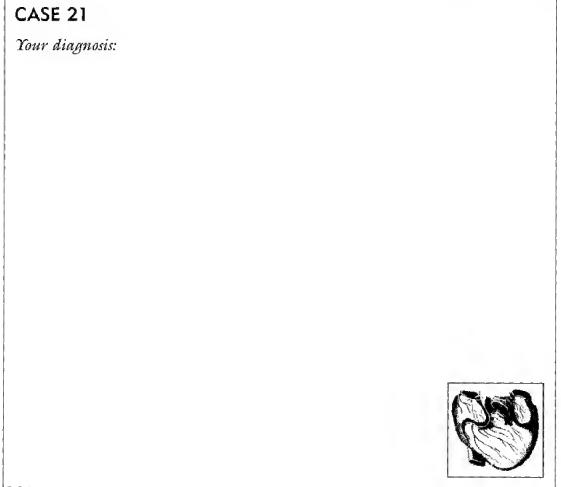


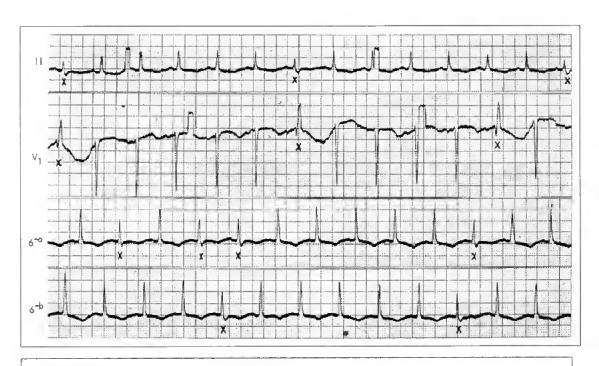




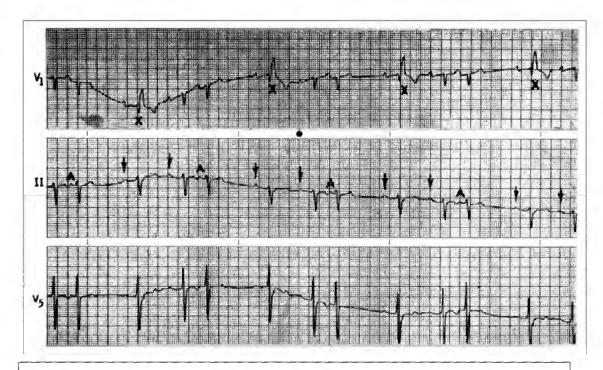




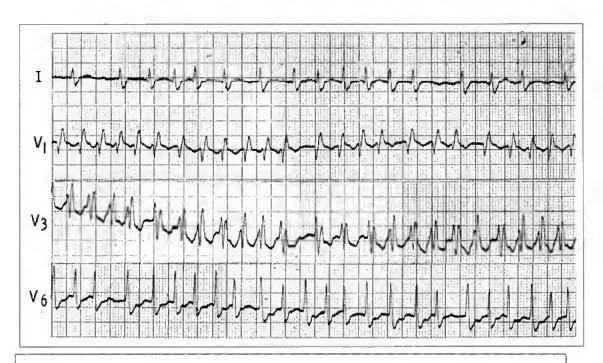




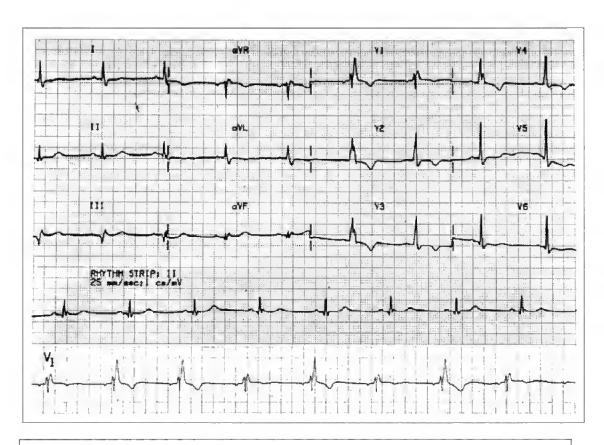


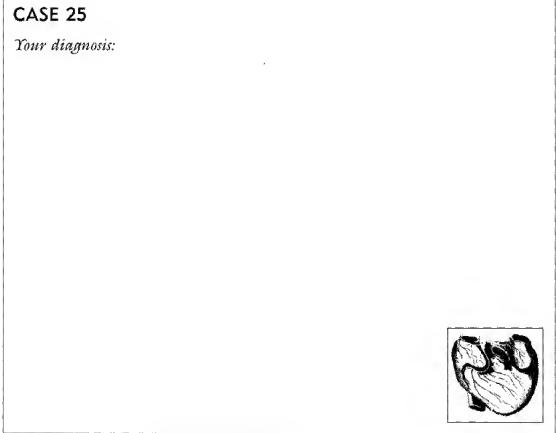


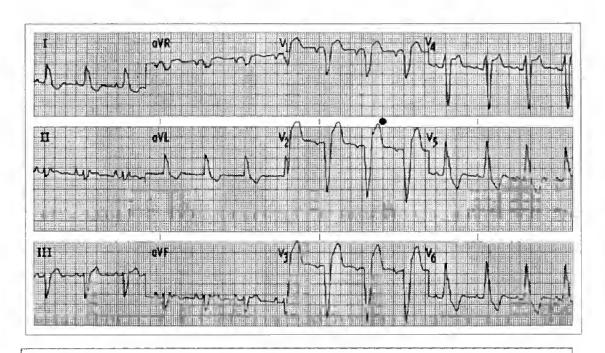




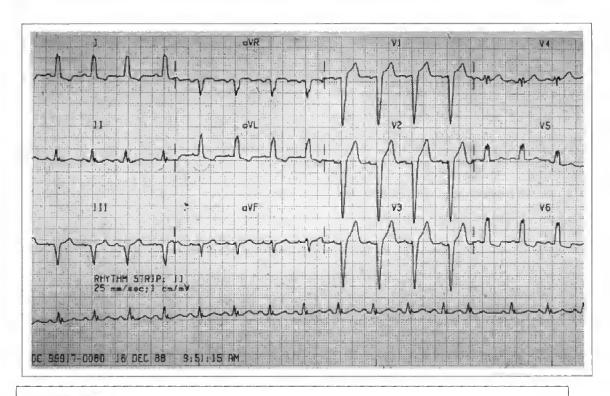




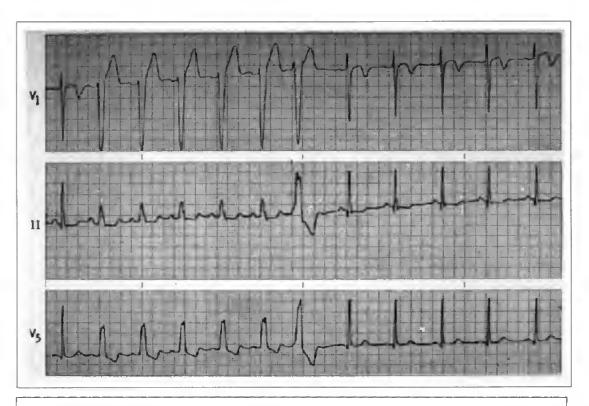










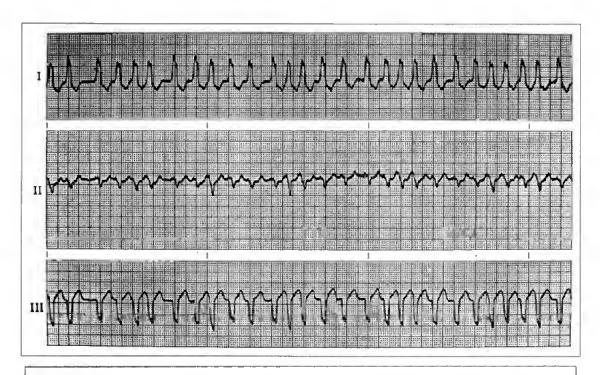




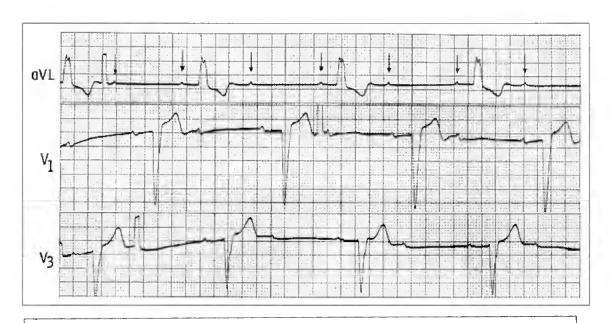






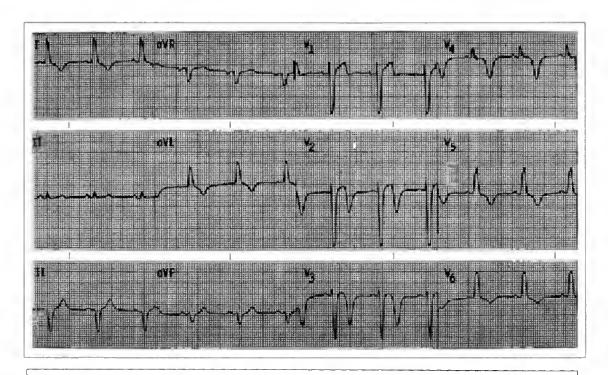




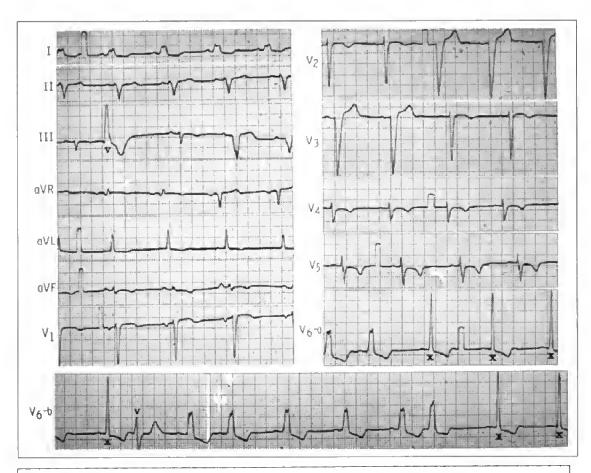






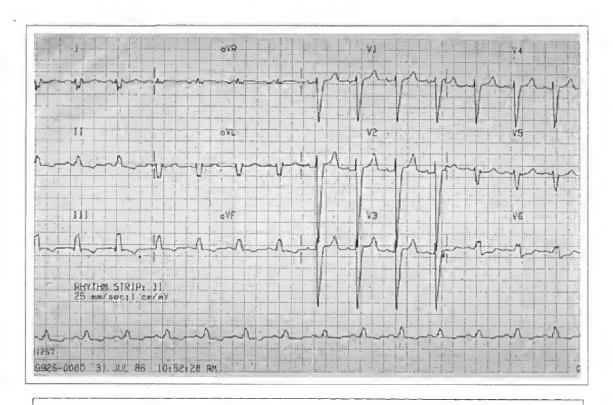




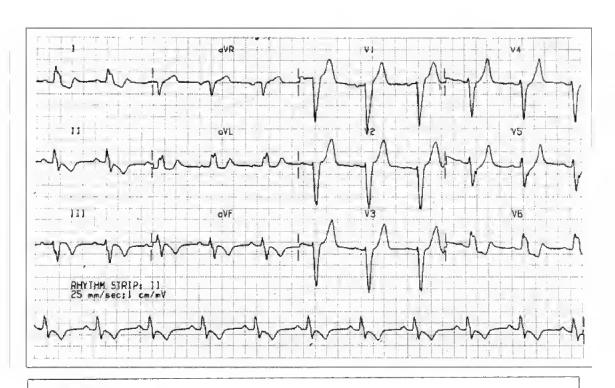


CASE 33



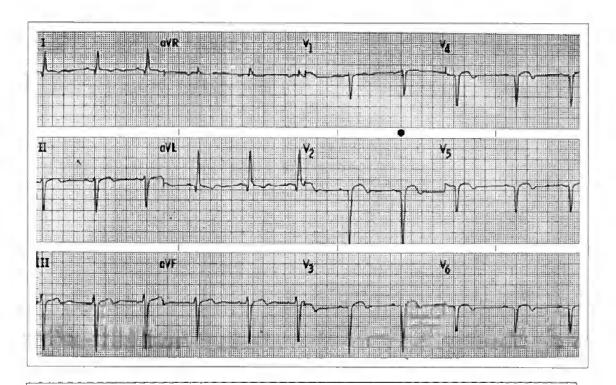




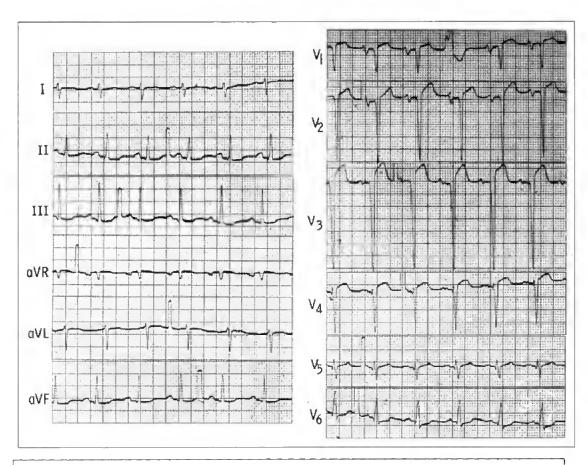




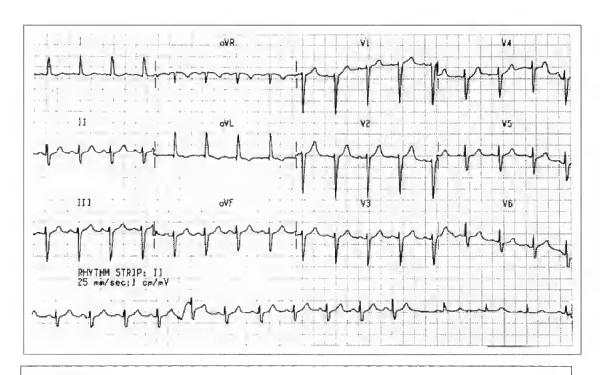




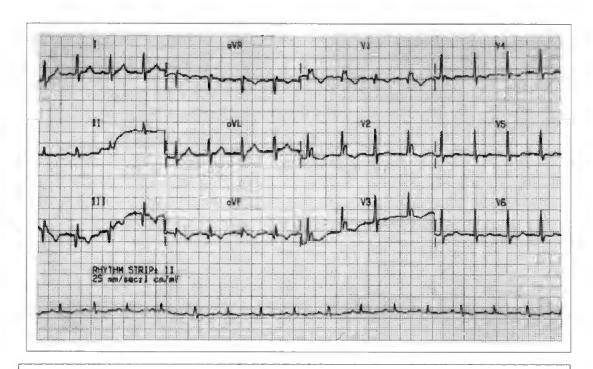




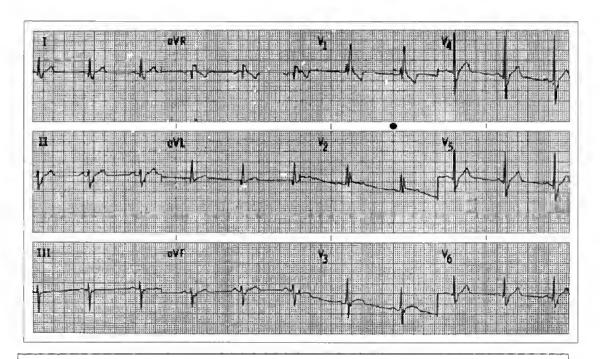




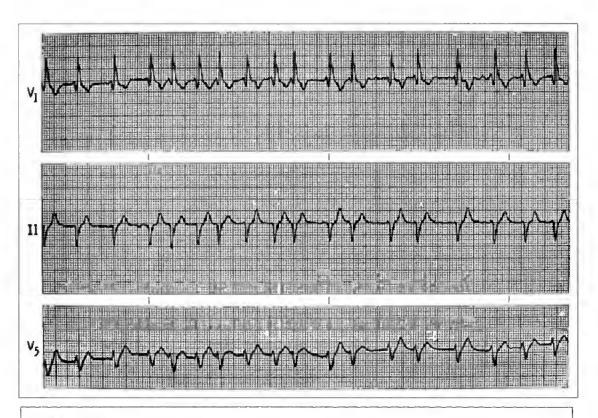




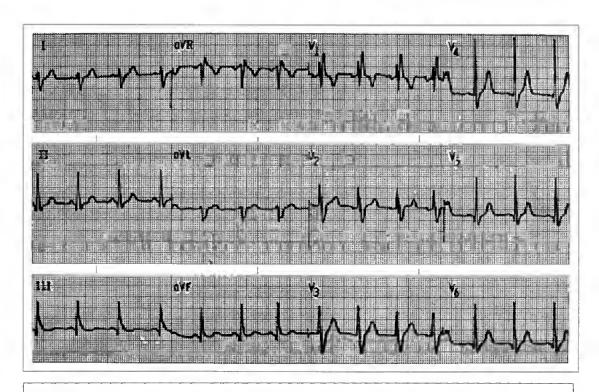




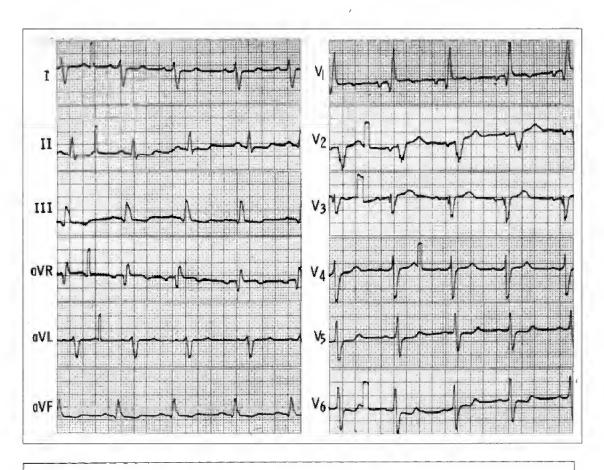






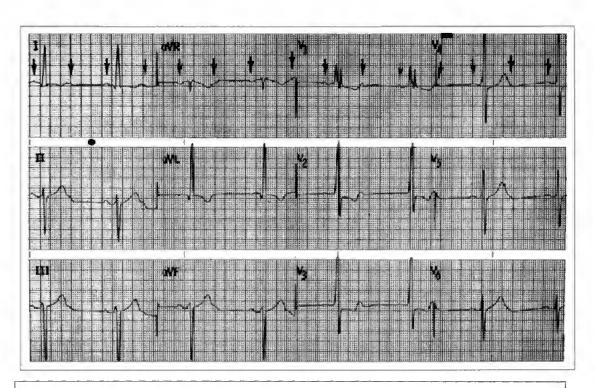




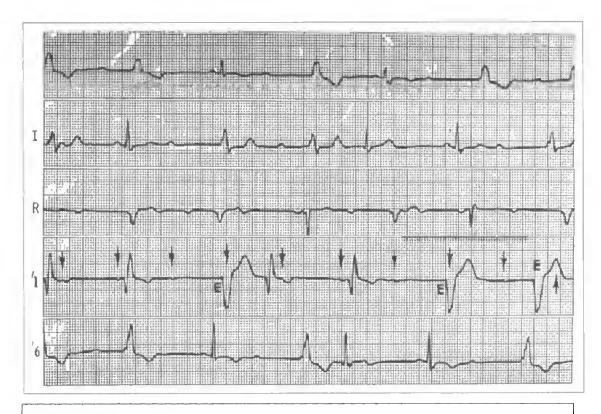




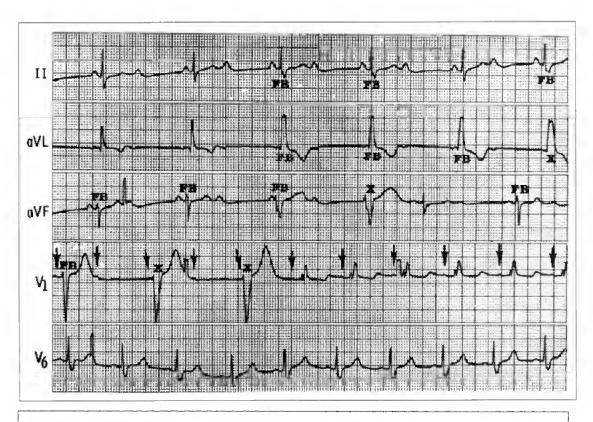




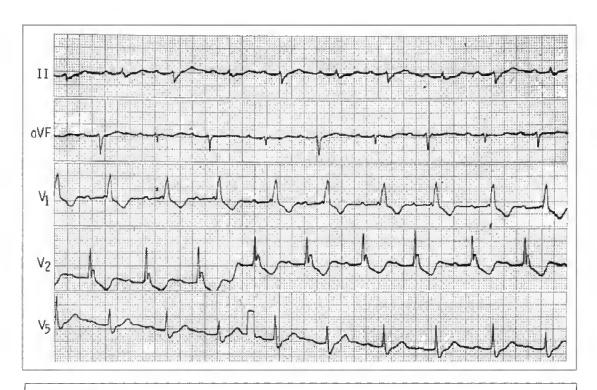




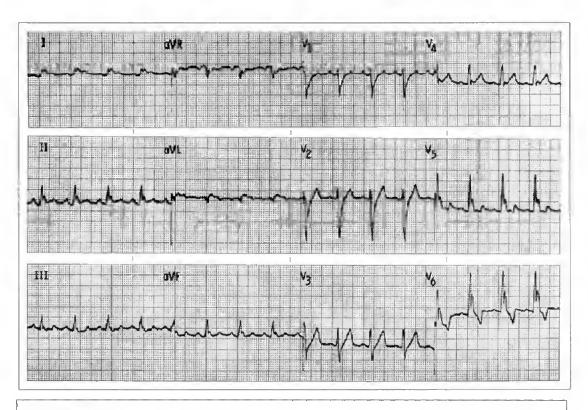




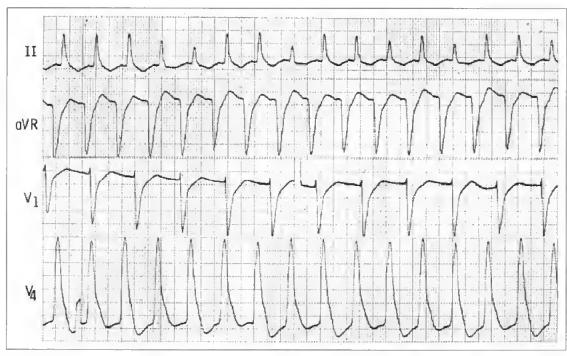


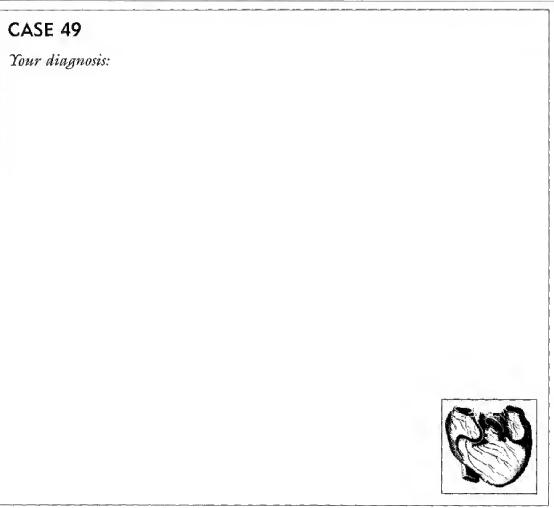


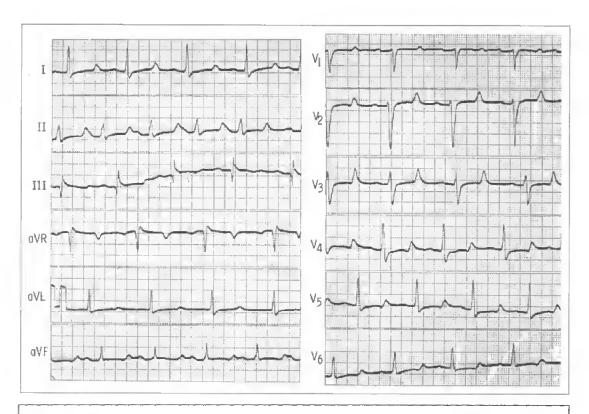




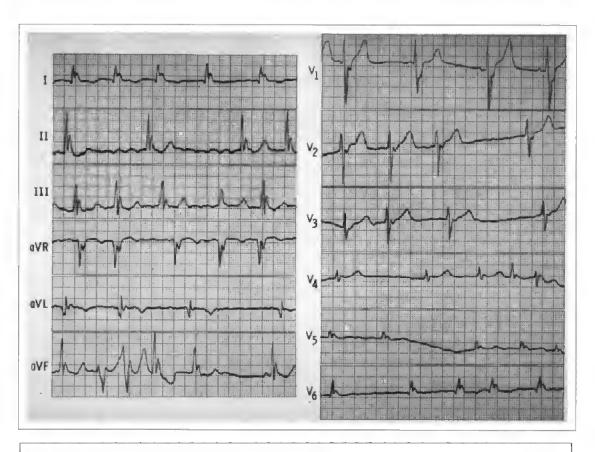




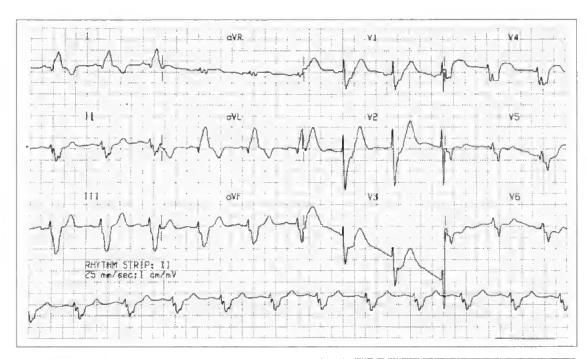


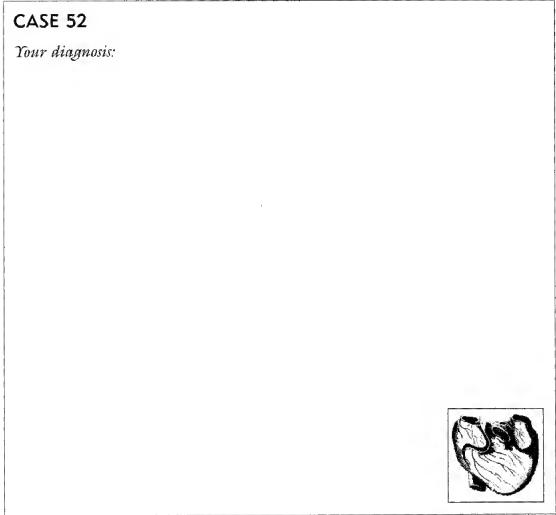


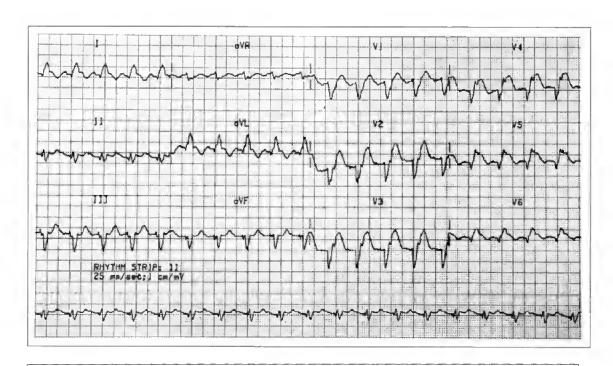


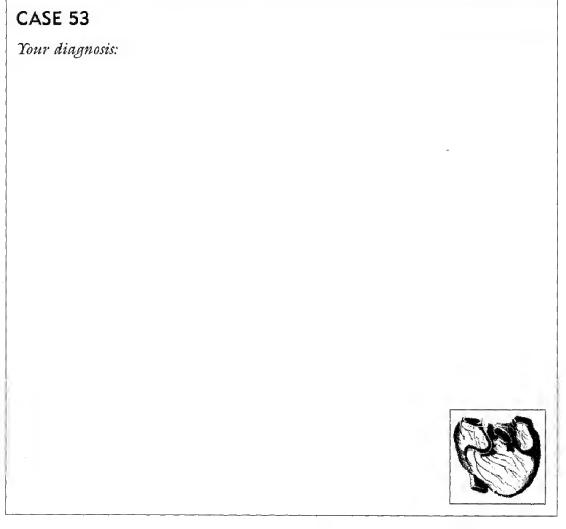


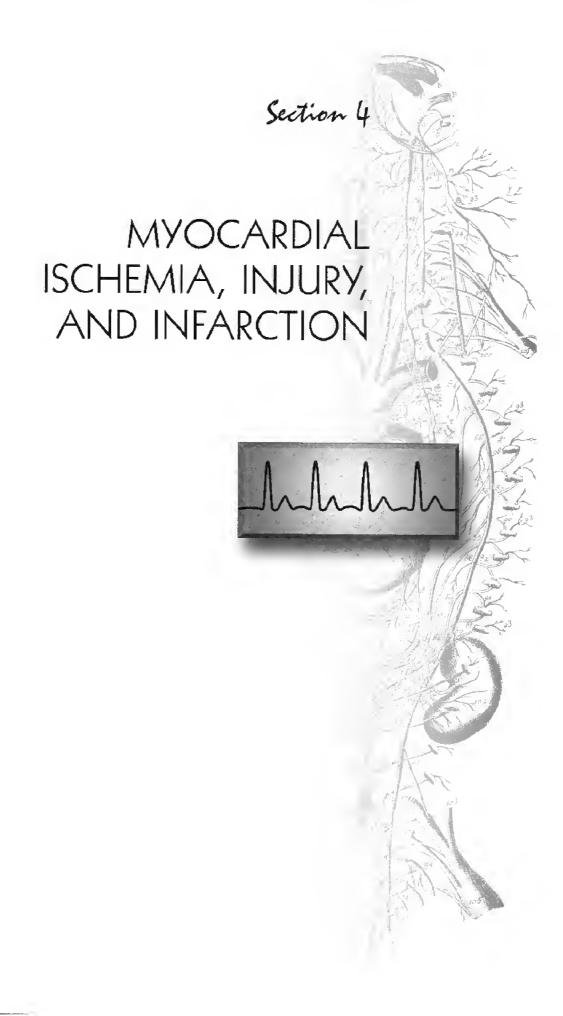


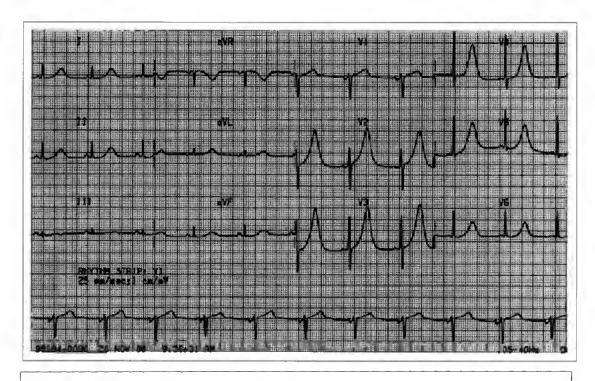




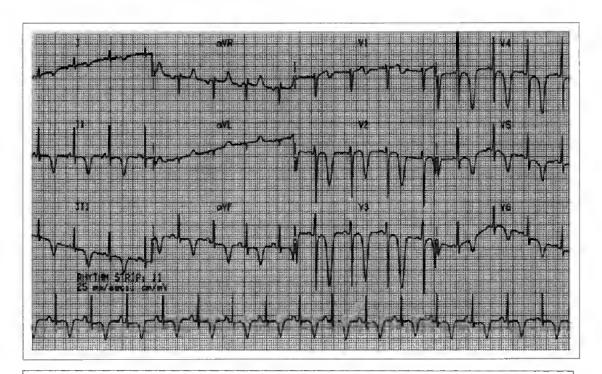




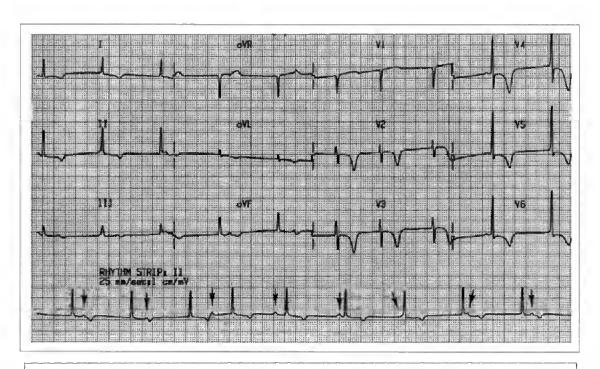




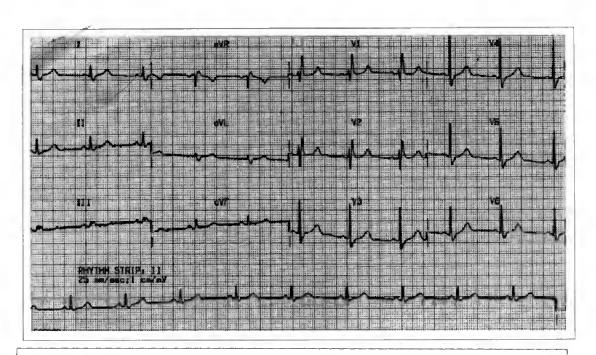




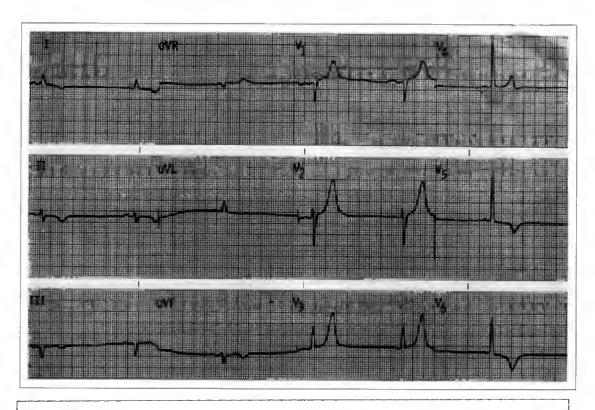




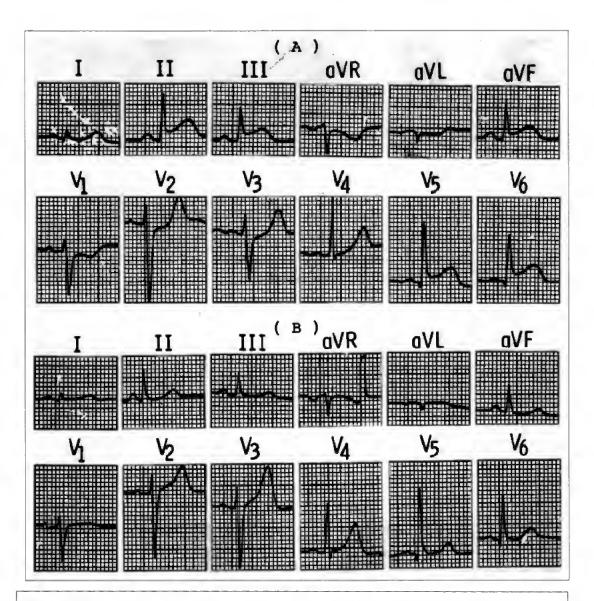






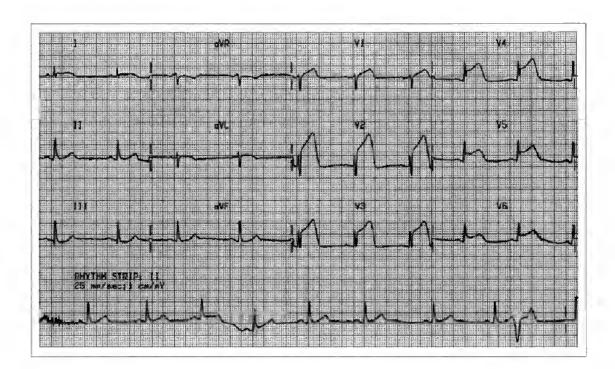




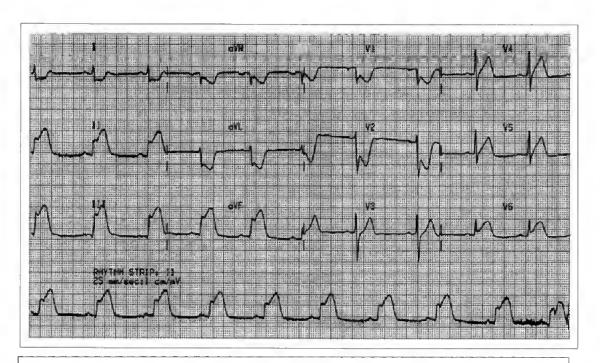


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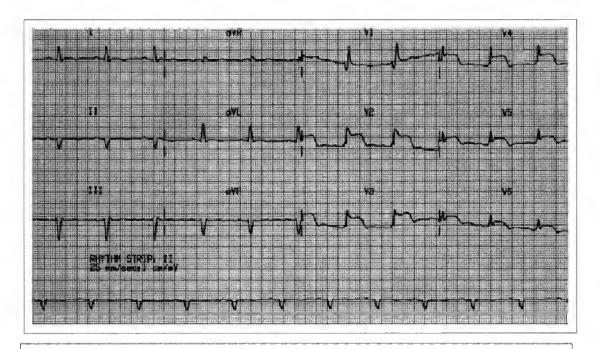




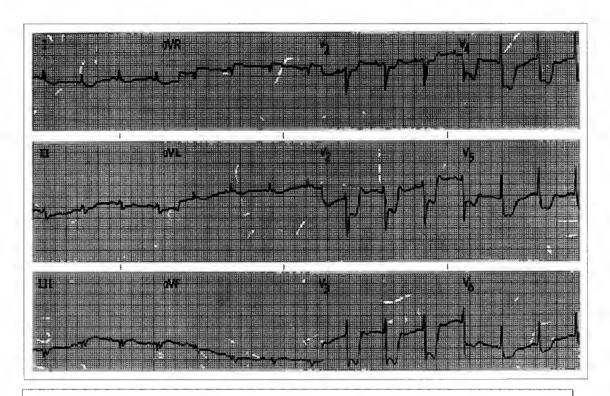




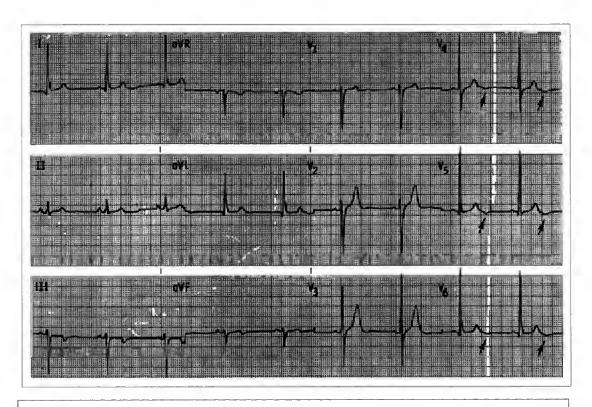




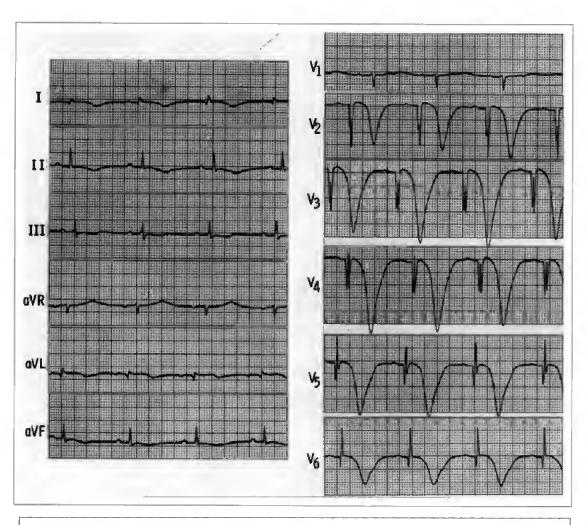




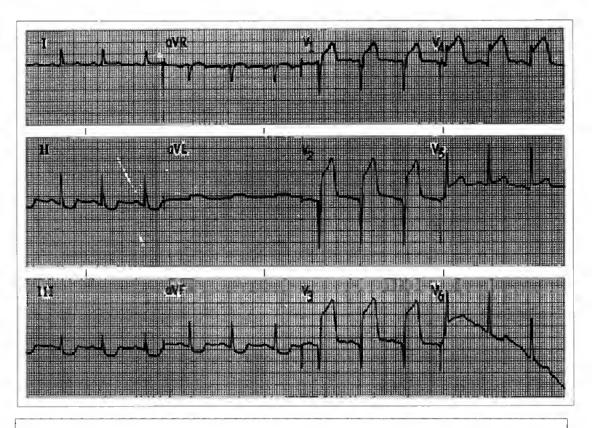




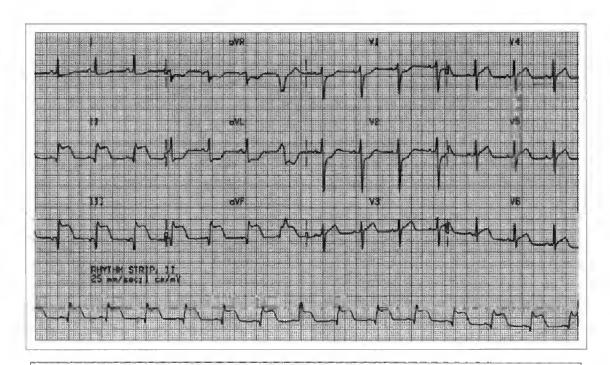




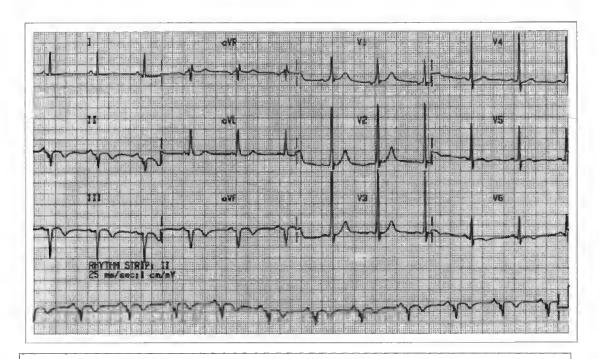




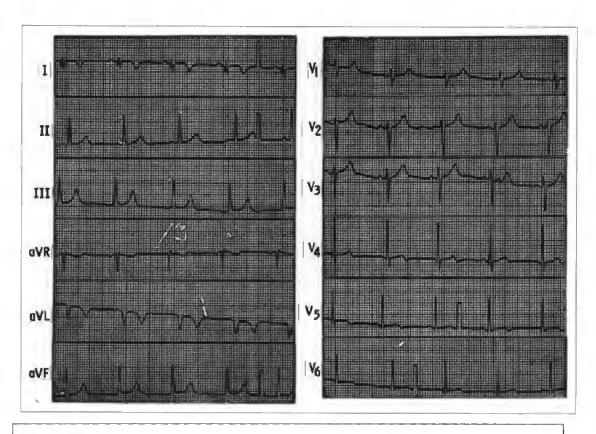




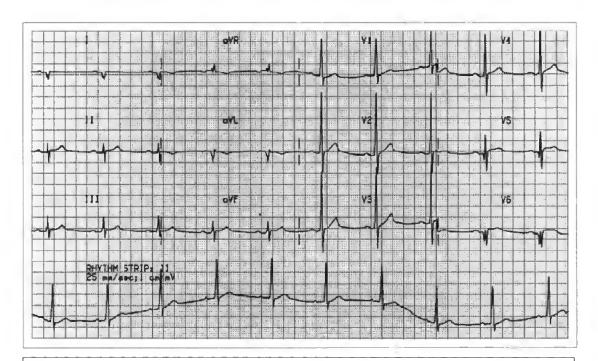




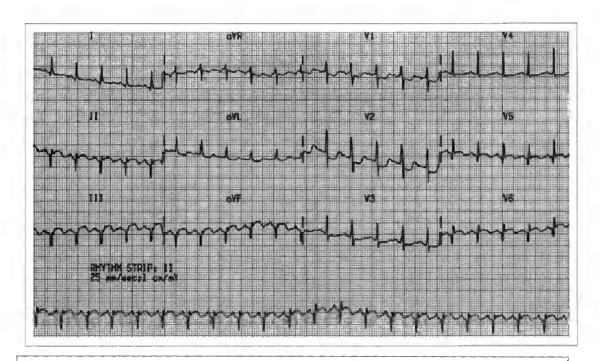




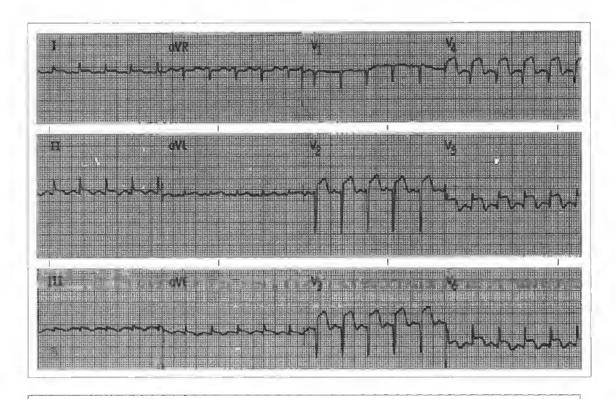




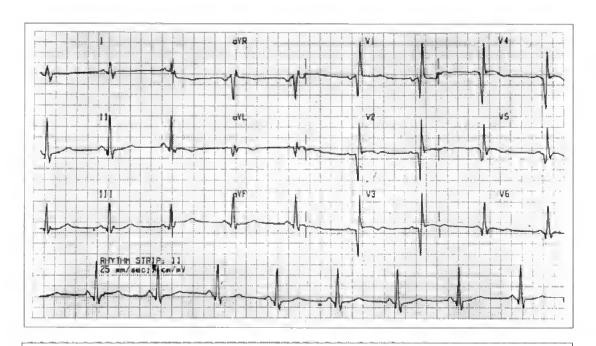




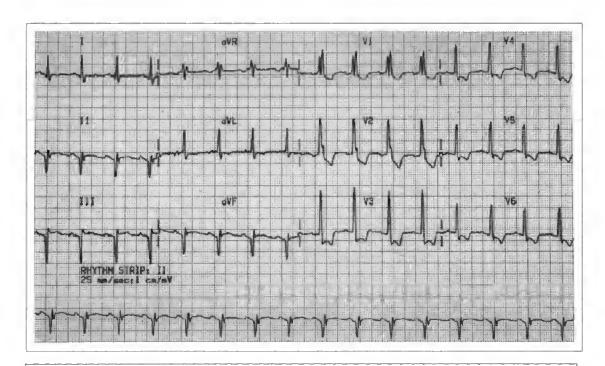




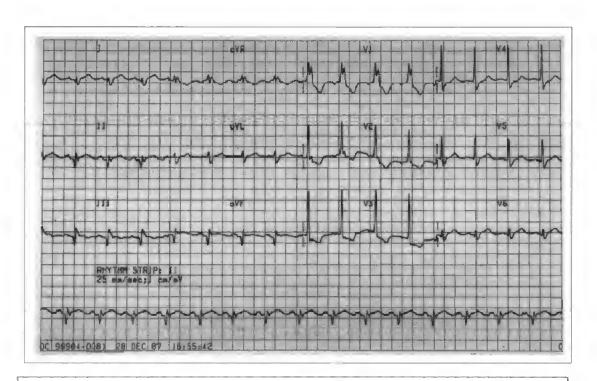




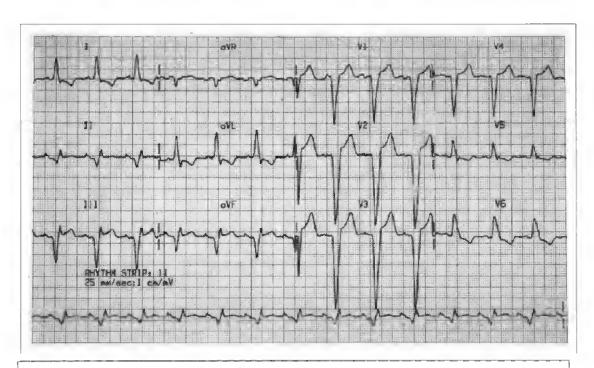




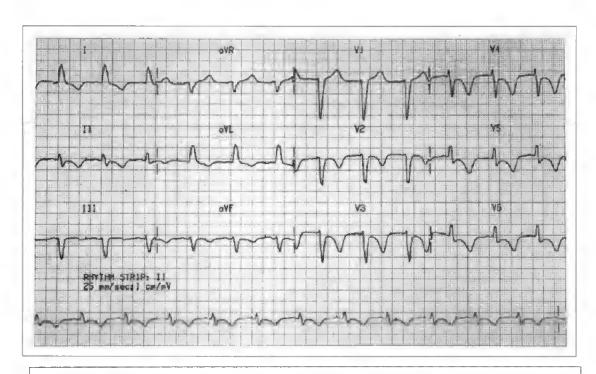




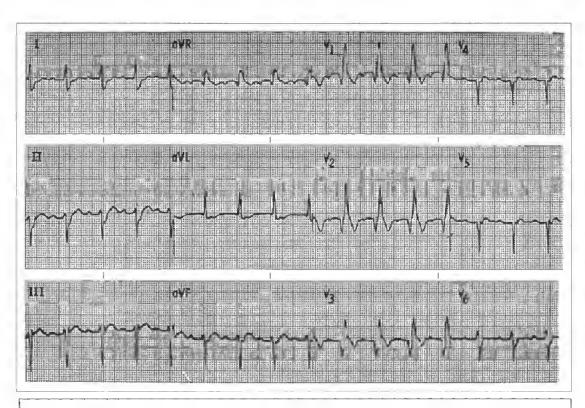




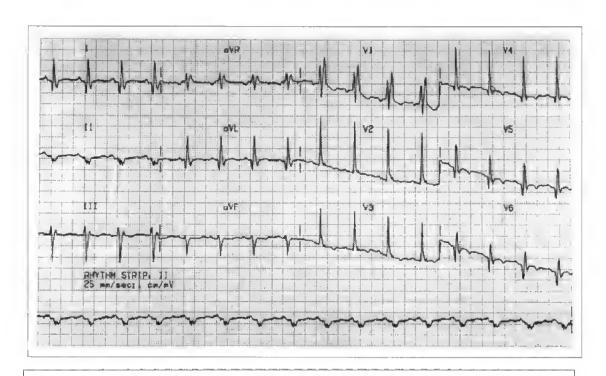




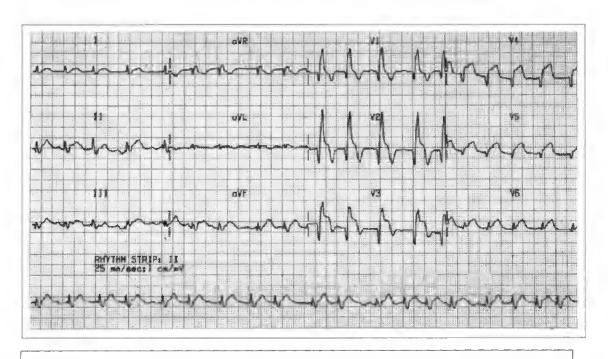




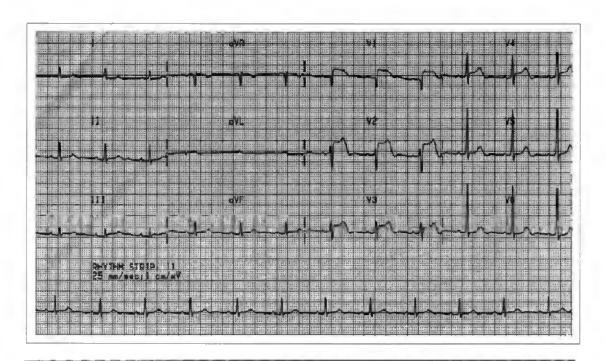




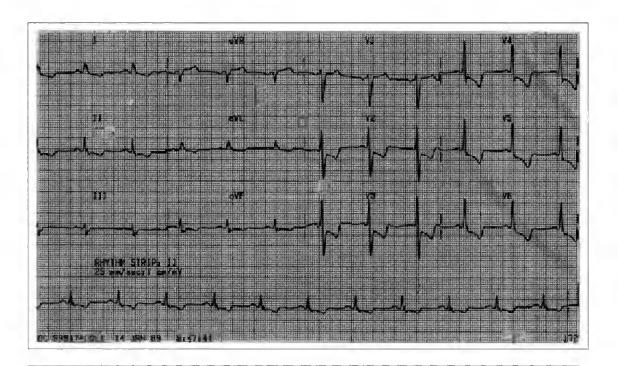




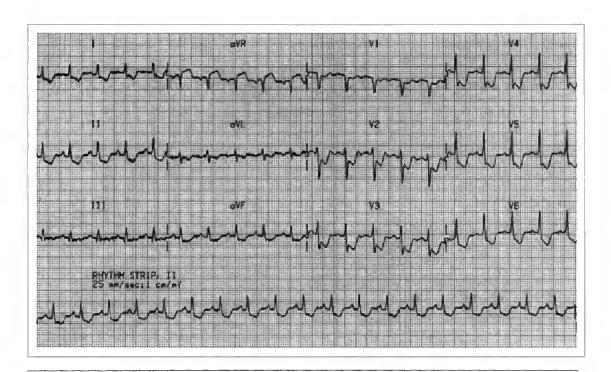




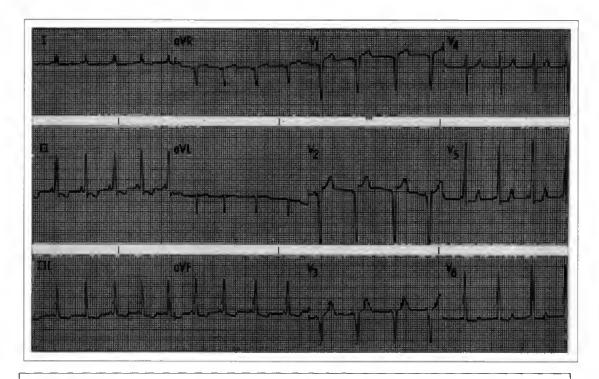




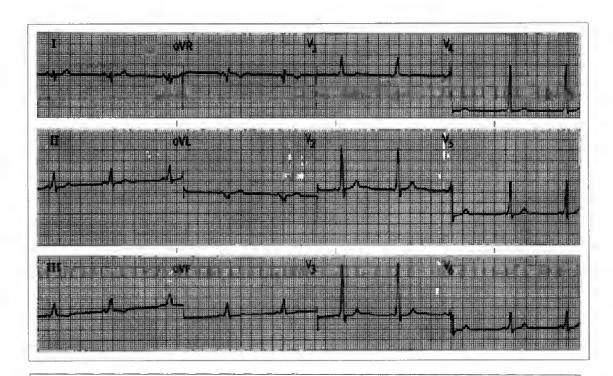




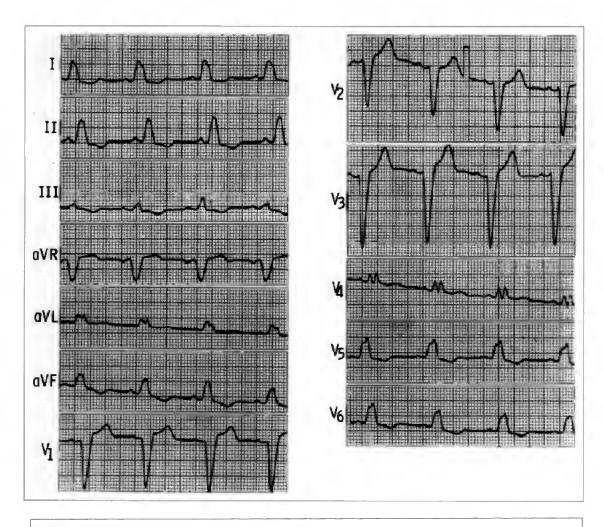




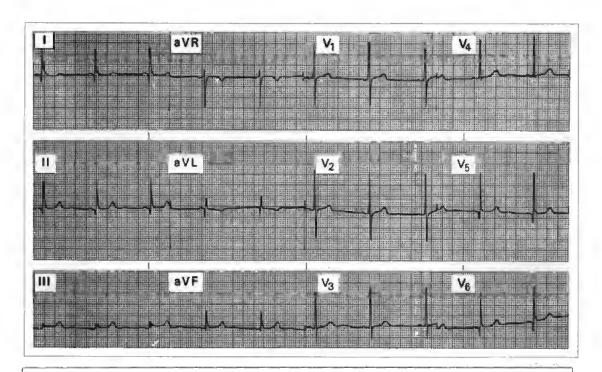




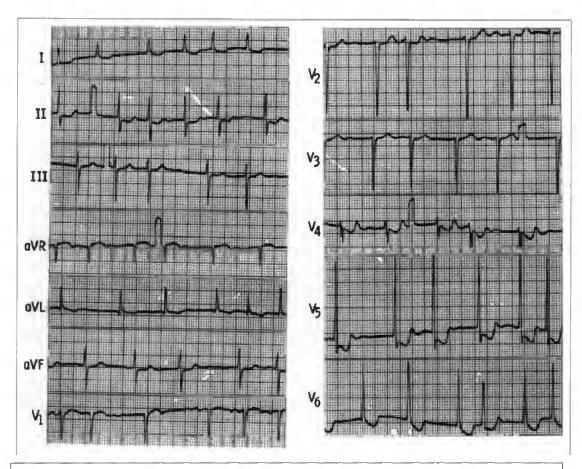






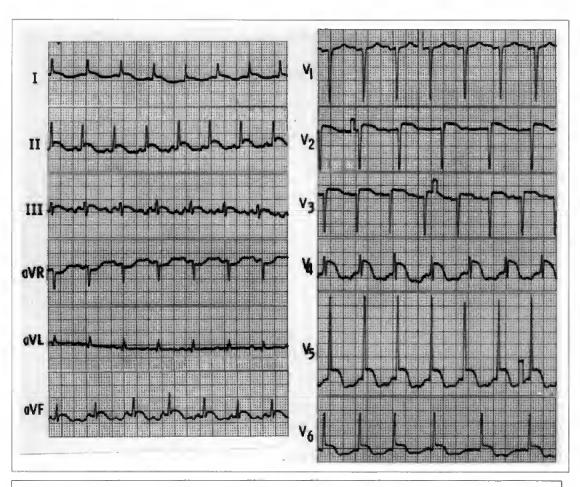






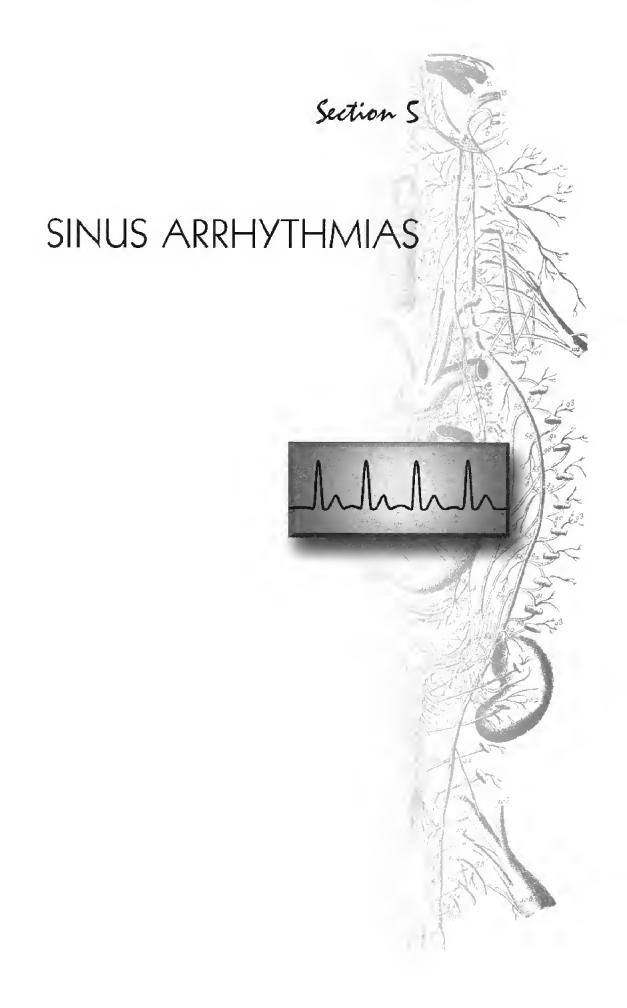
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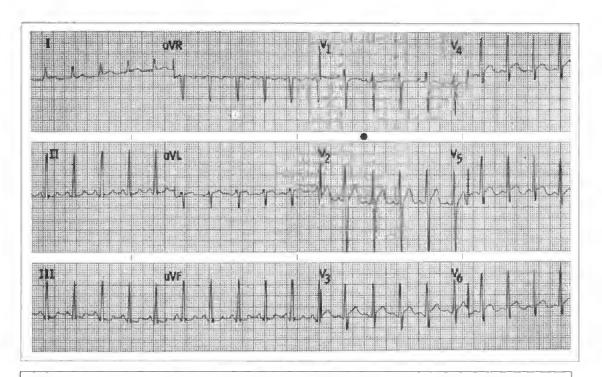




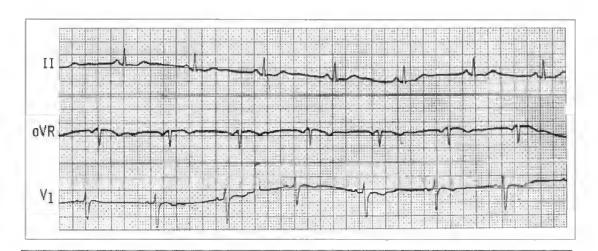


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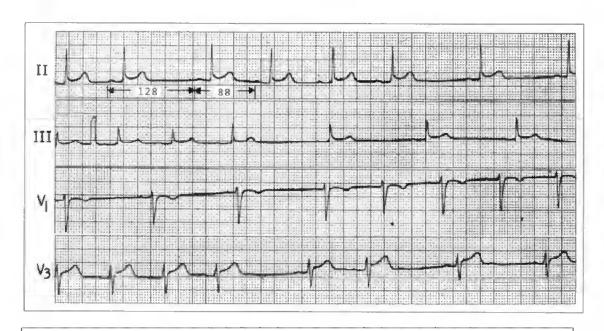




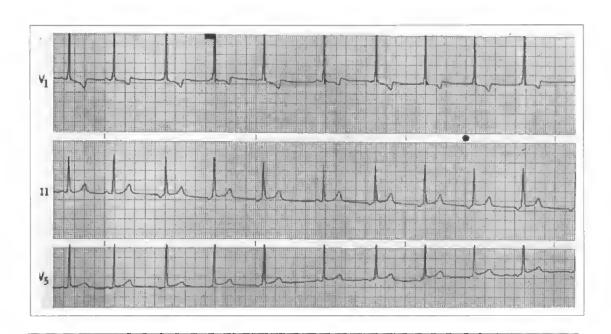




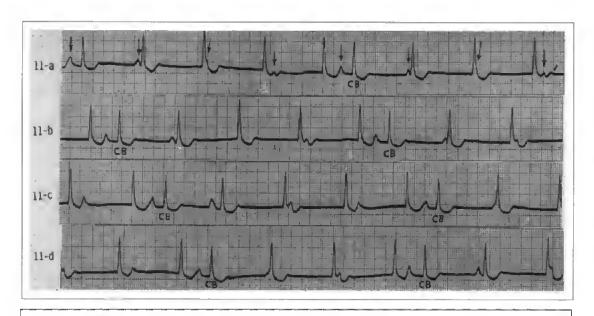




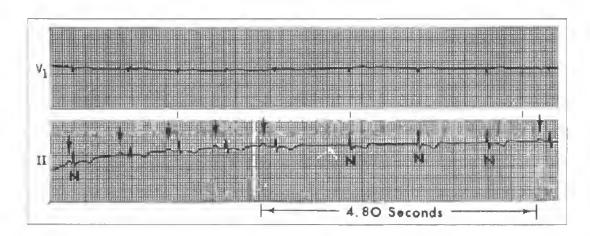




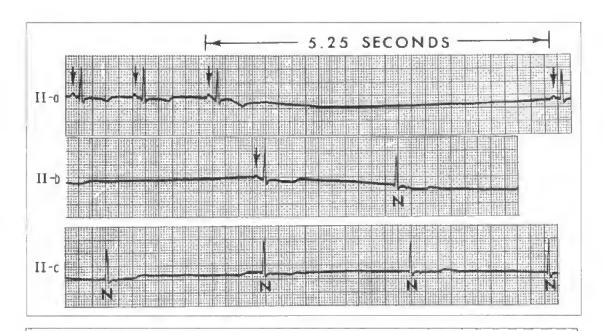




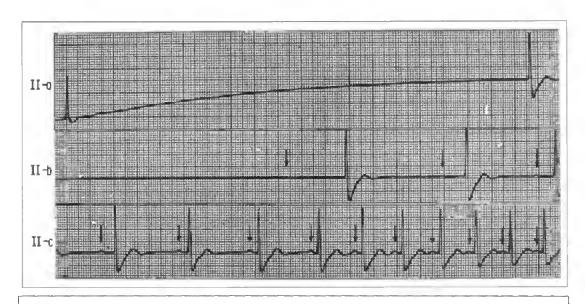




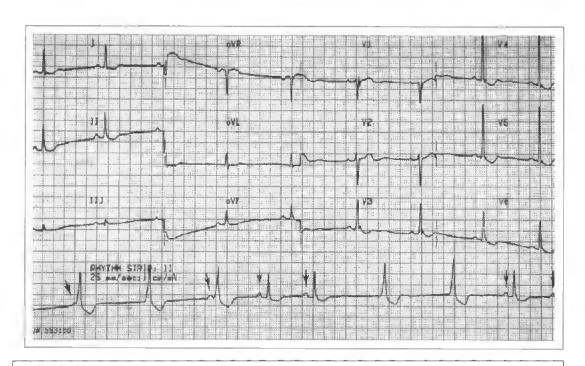




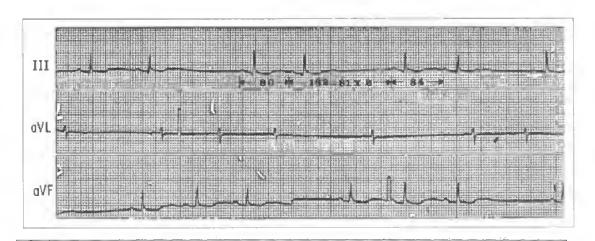




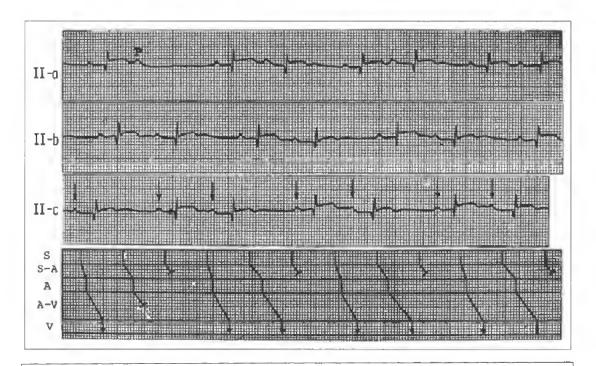




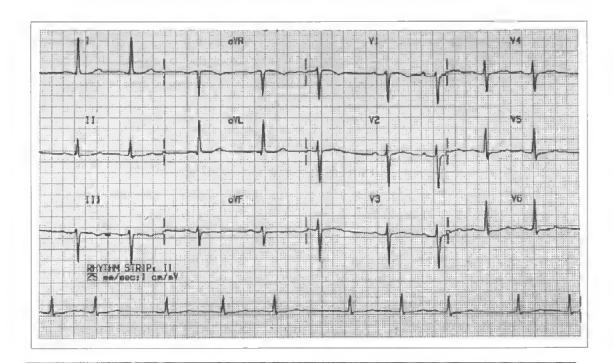




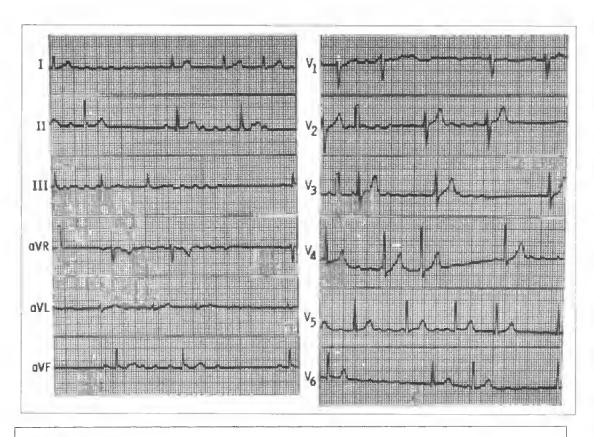




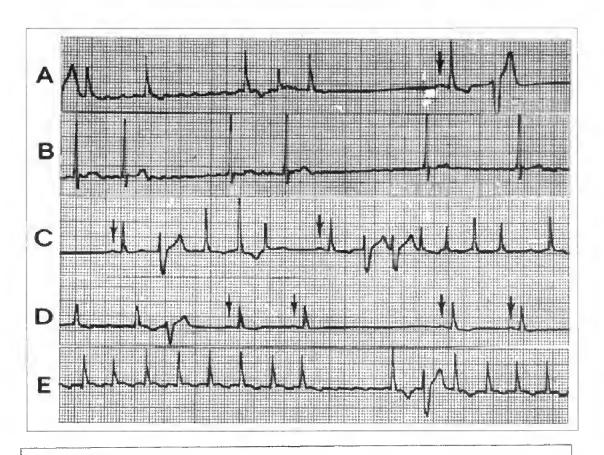




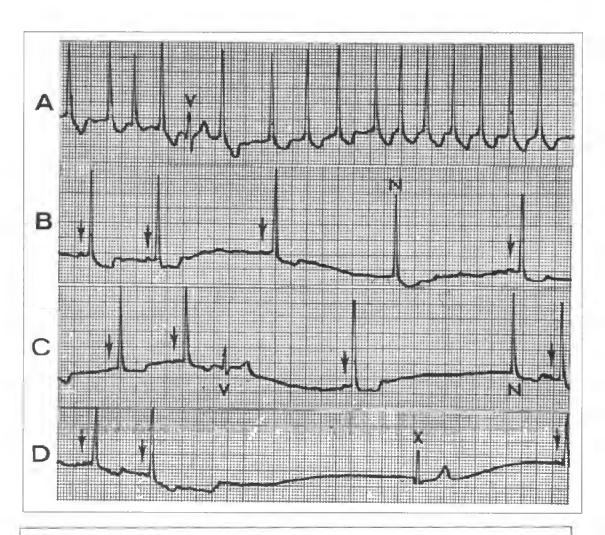




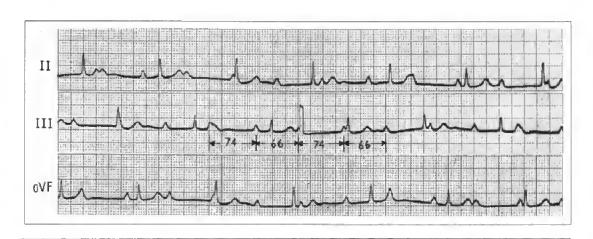






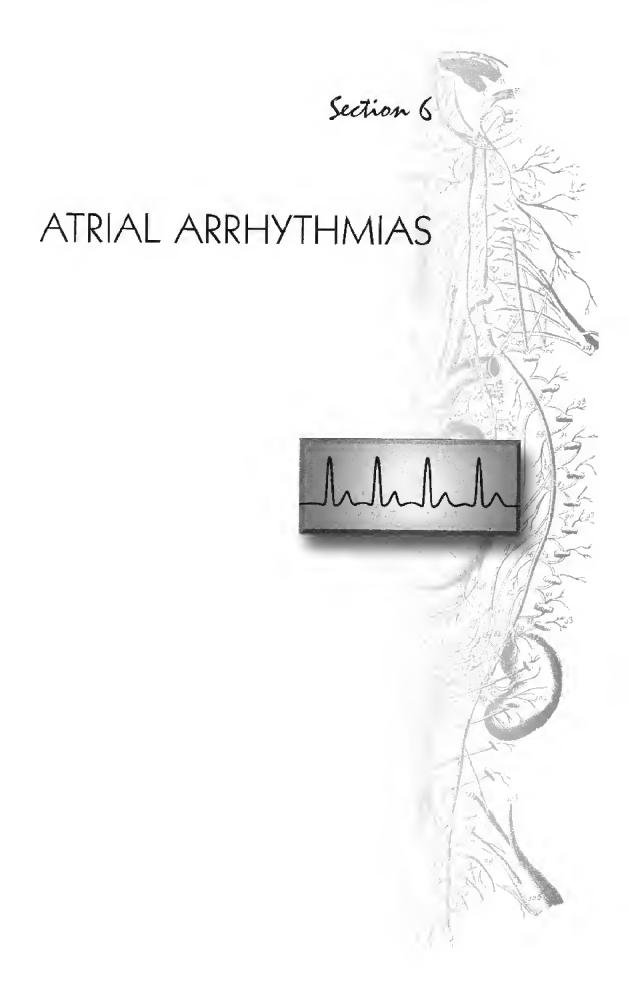


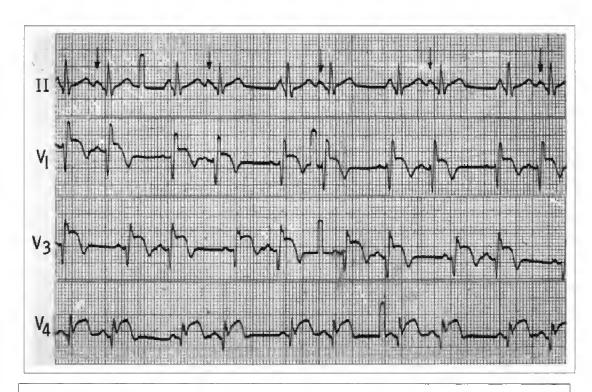






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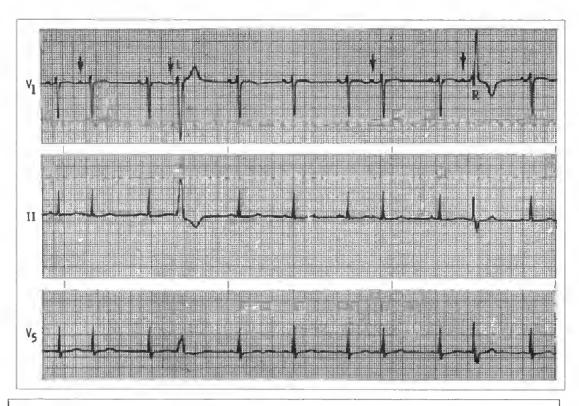




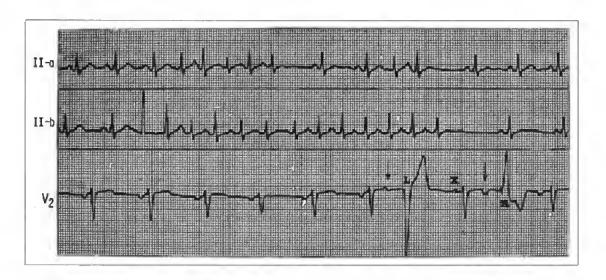




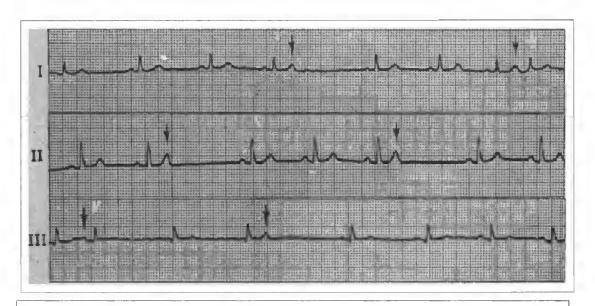




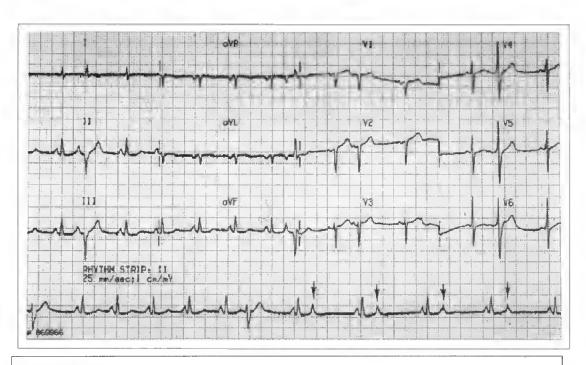




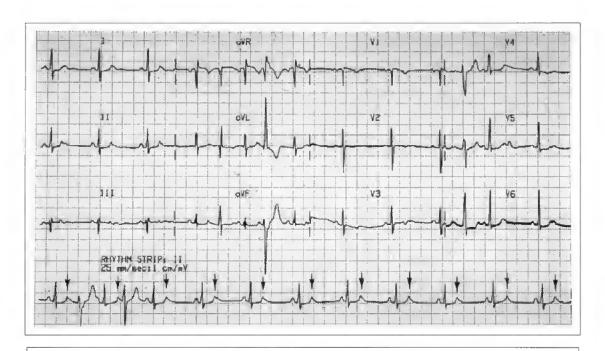




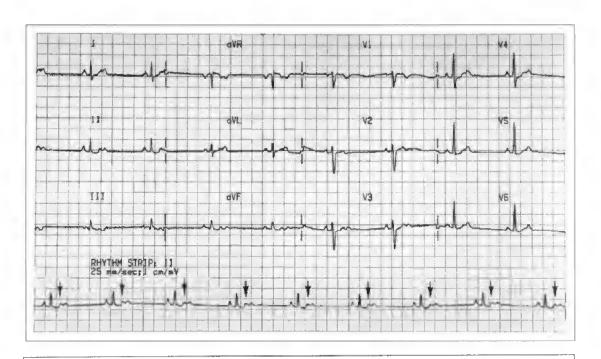




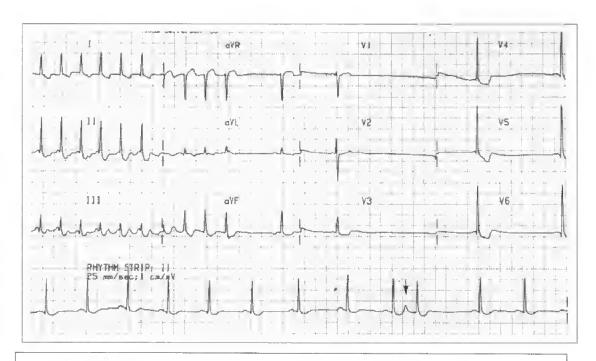




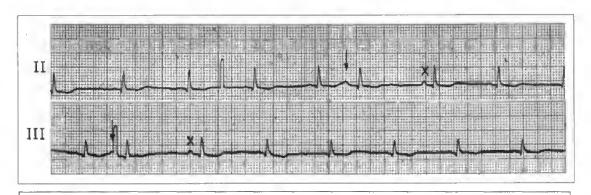




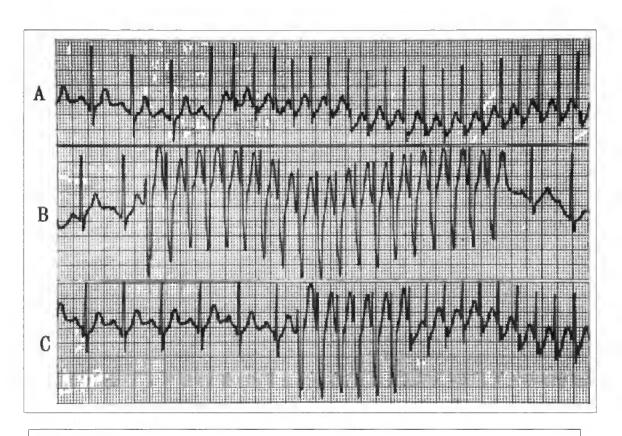








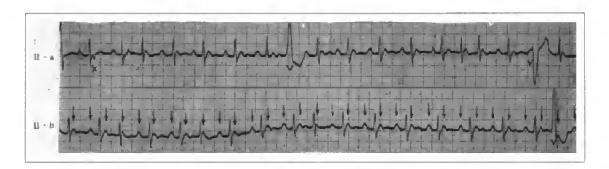






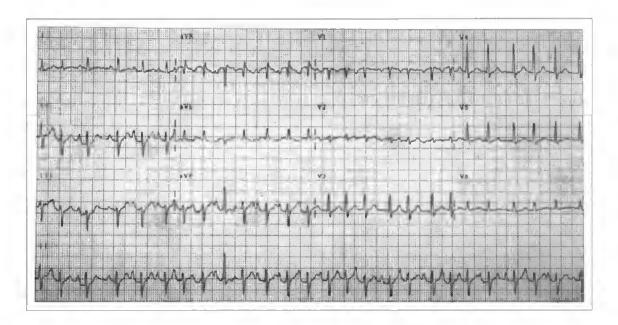




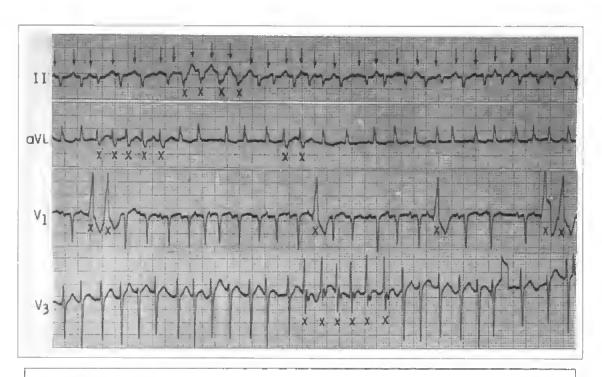


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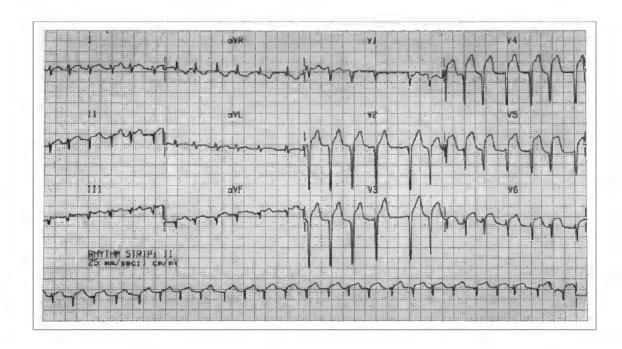




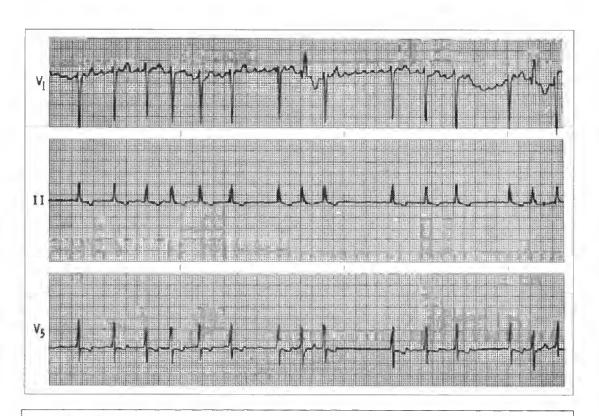




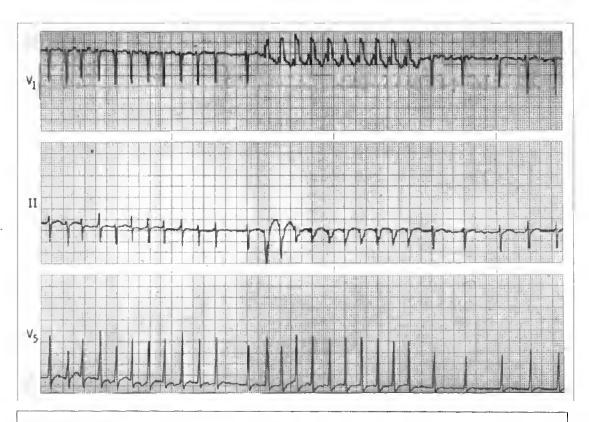




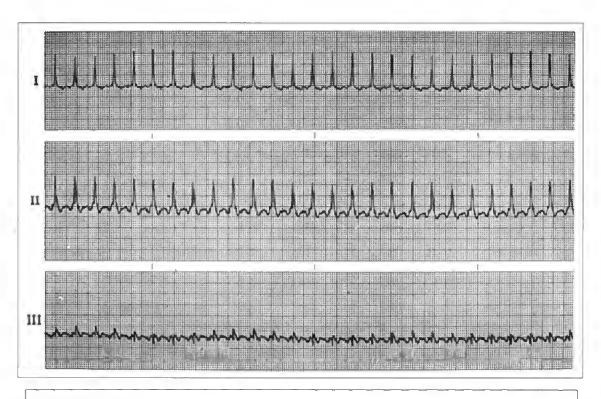






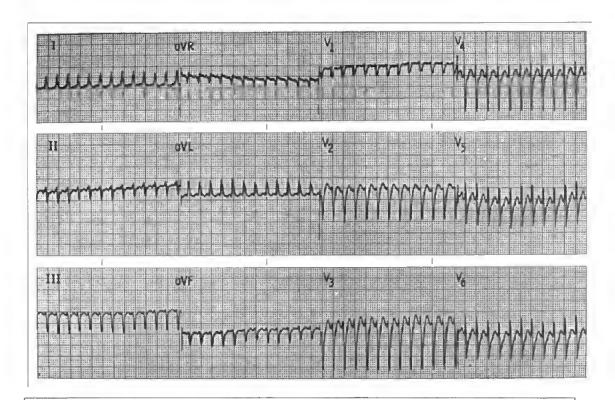




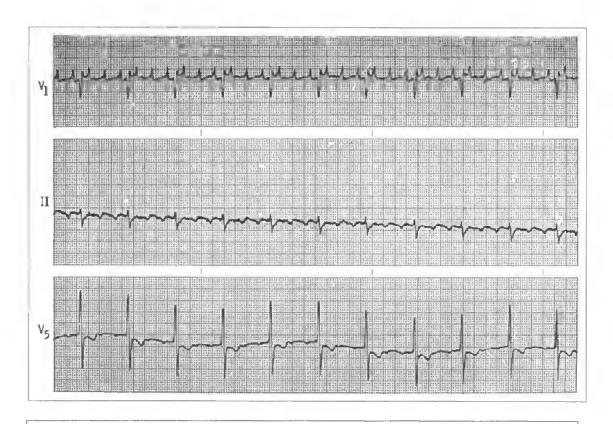




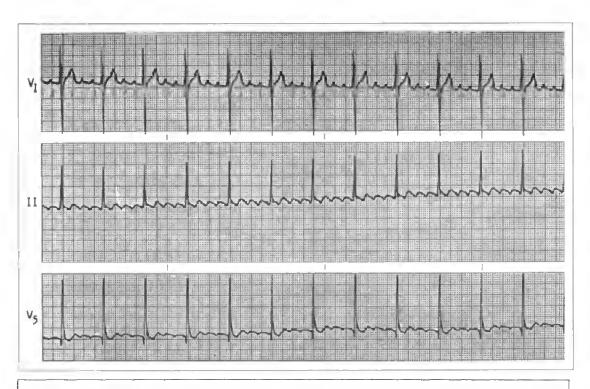




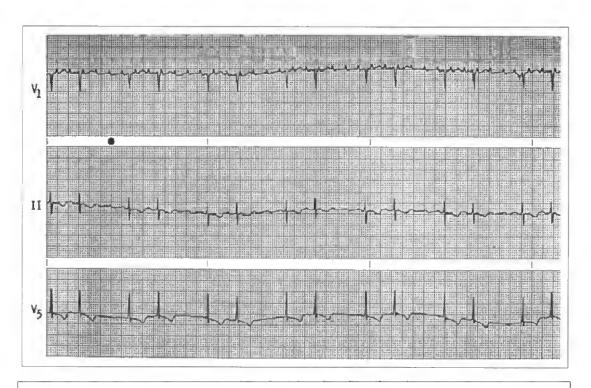




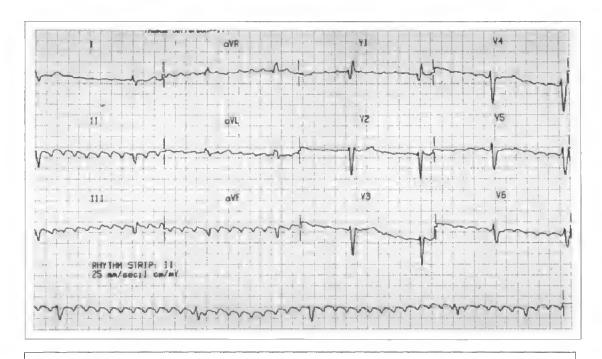






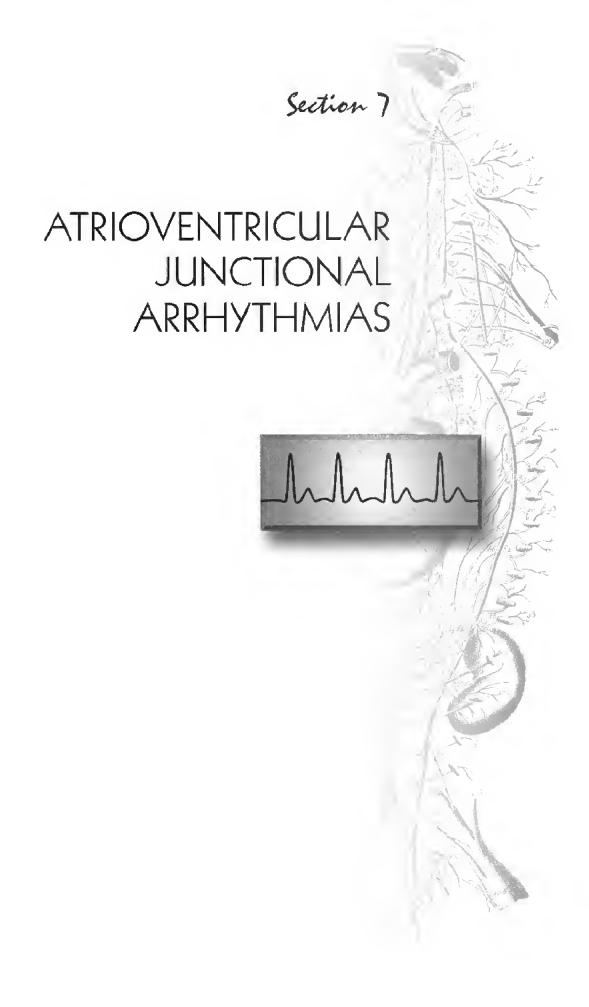


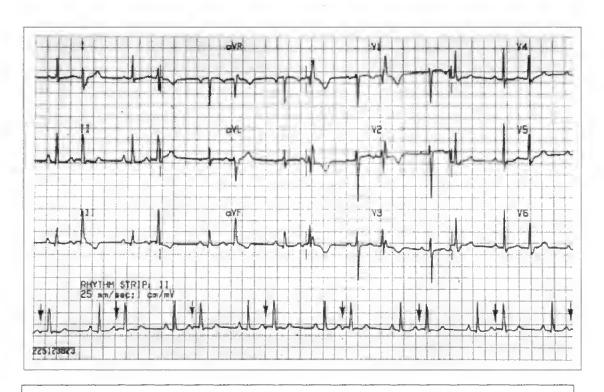




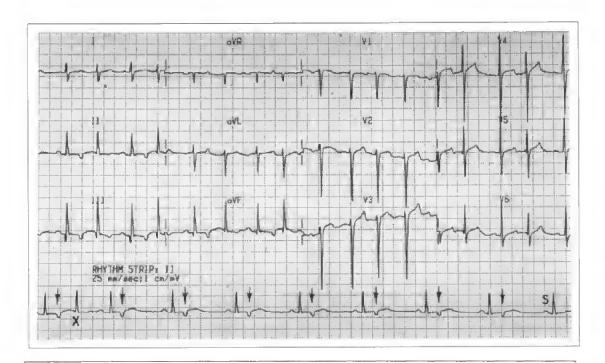


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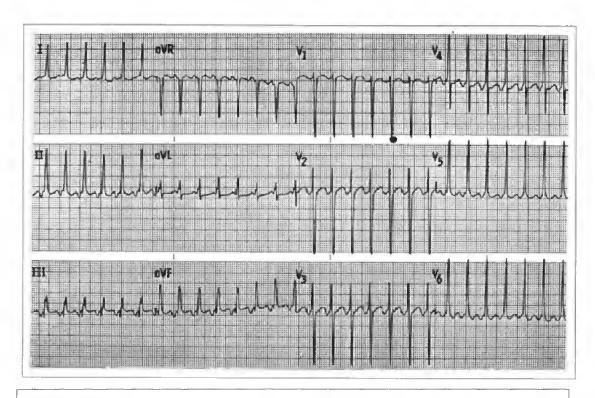




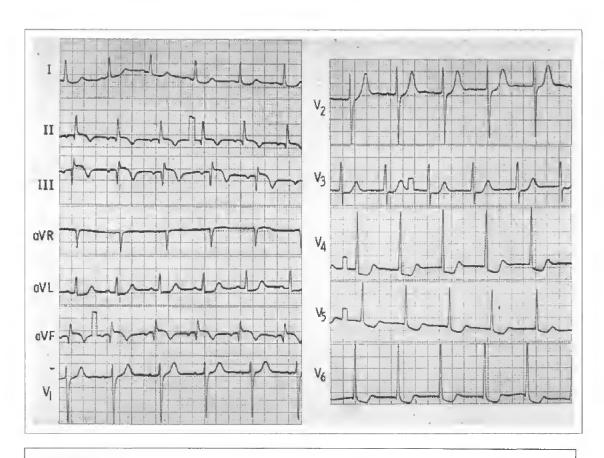




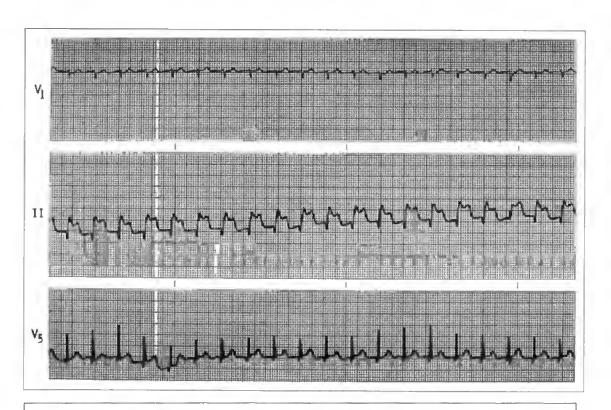




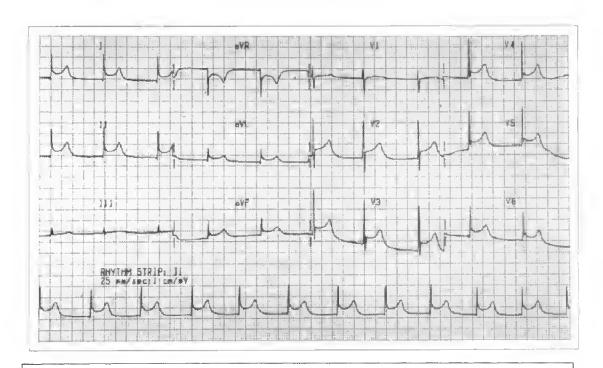




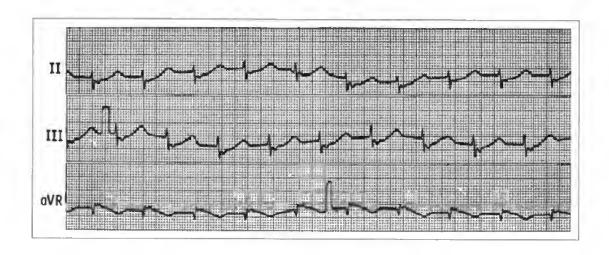




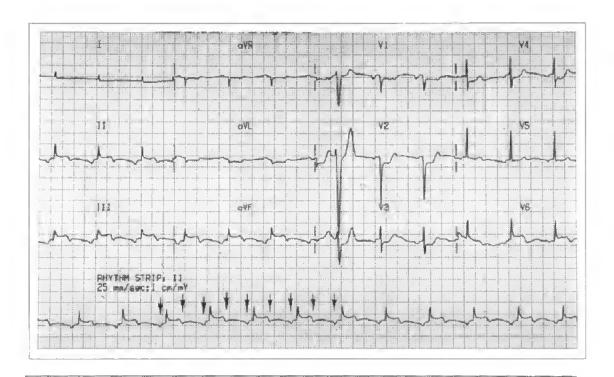




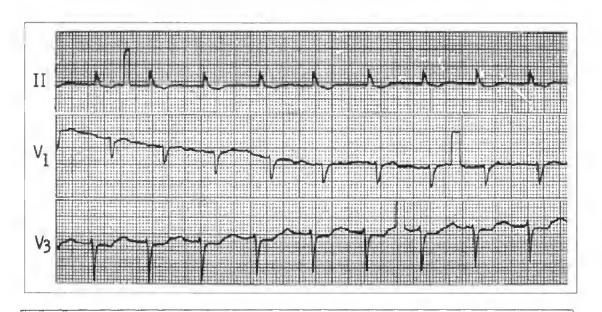




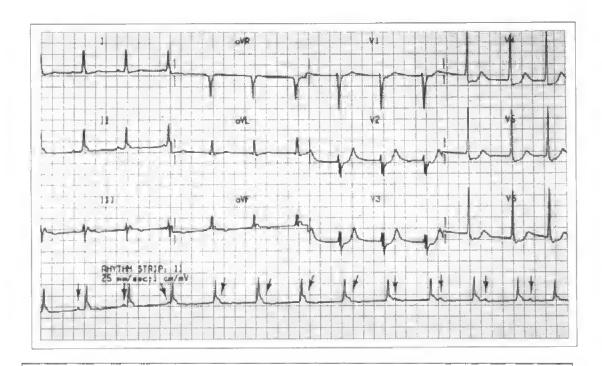




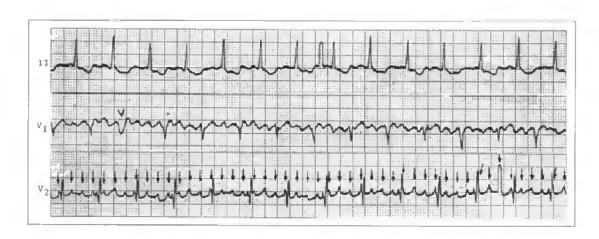




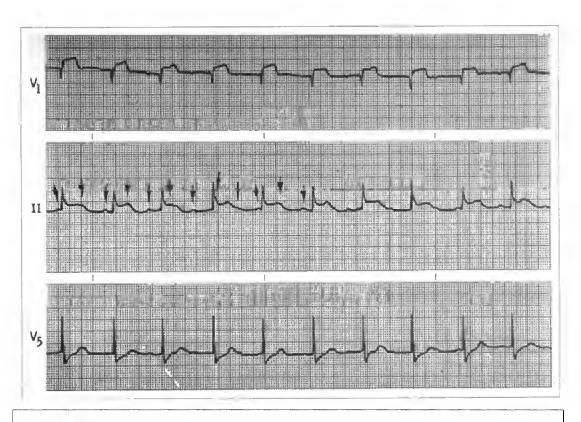




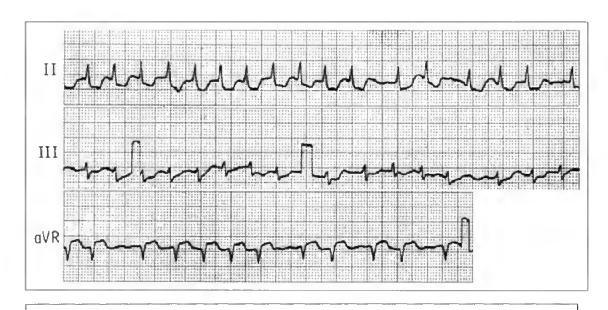




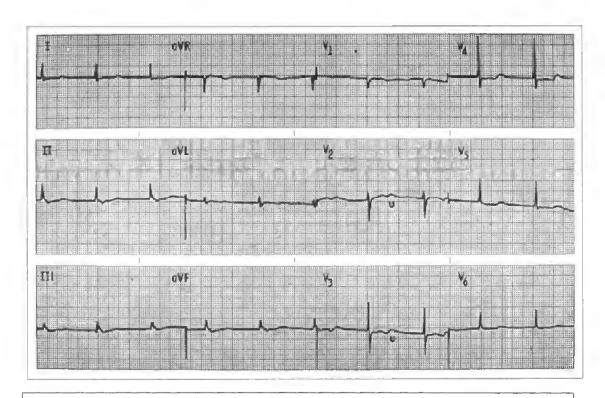




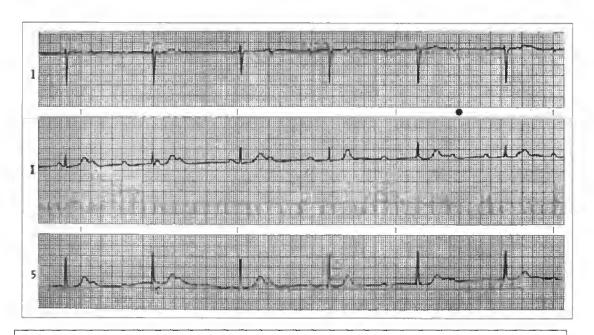




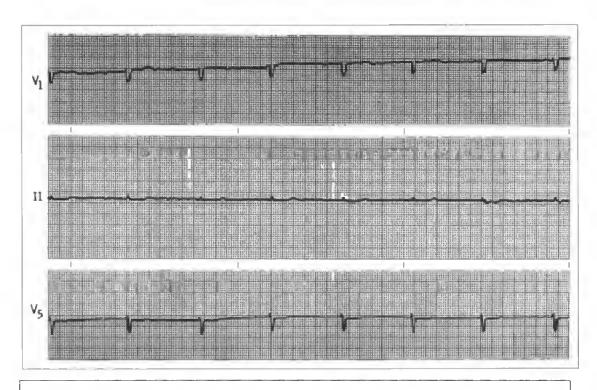




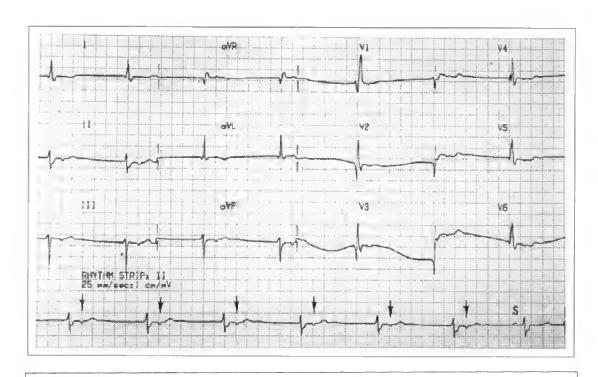




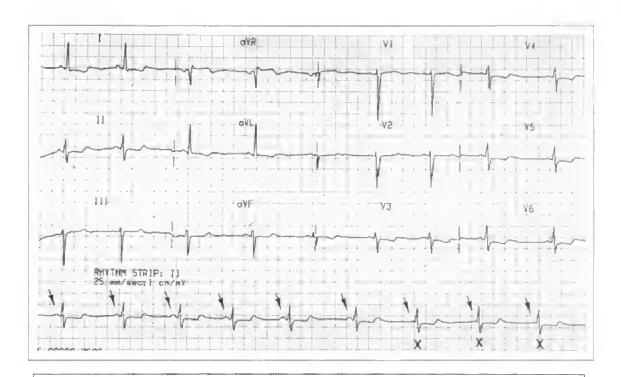




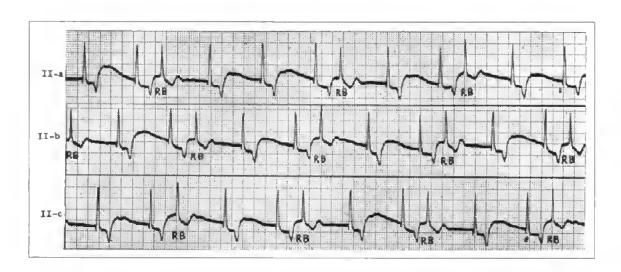




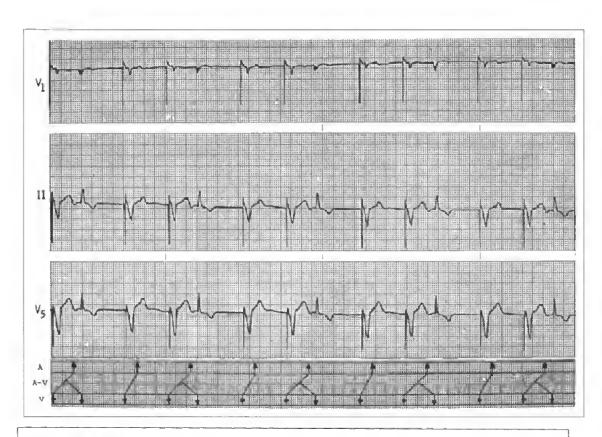




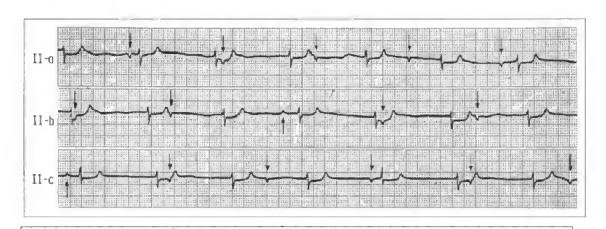




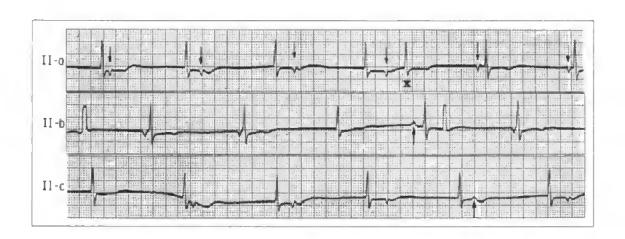




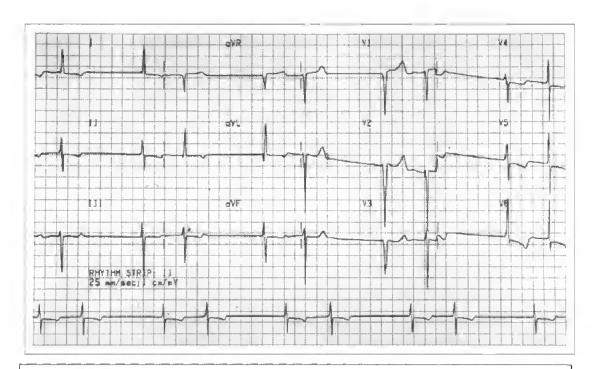




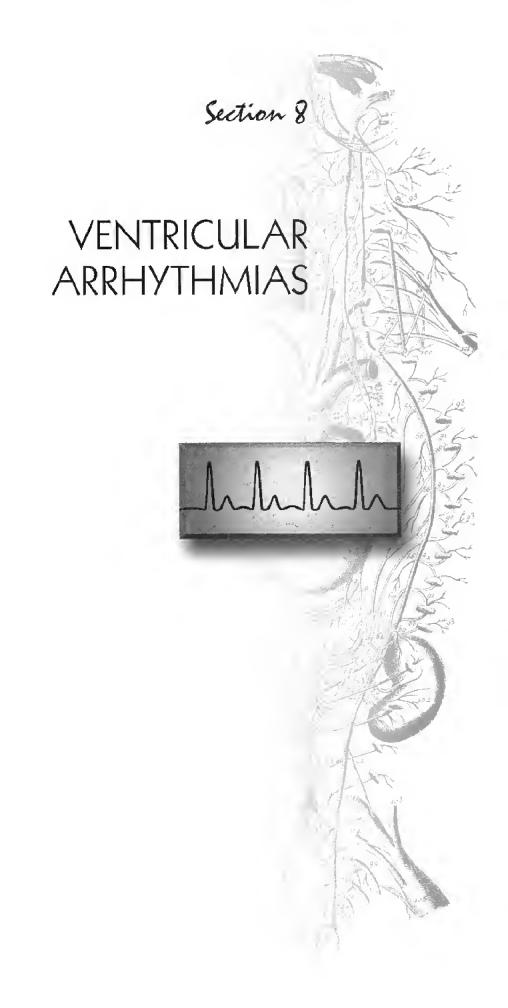


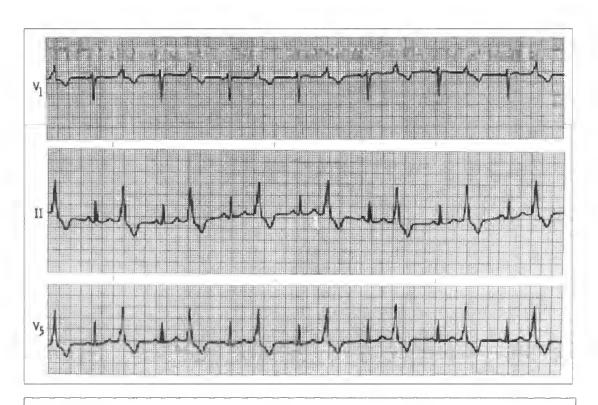




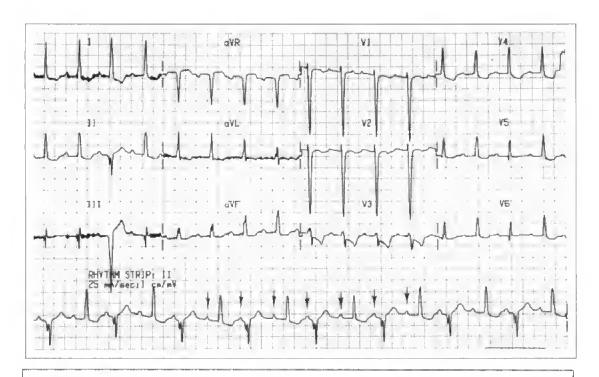




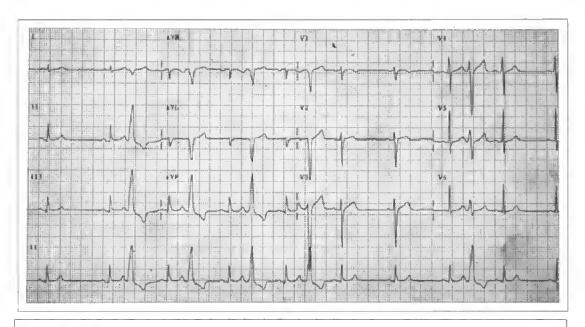




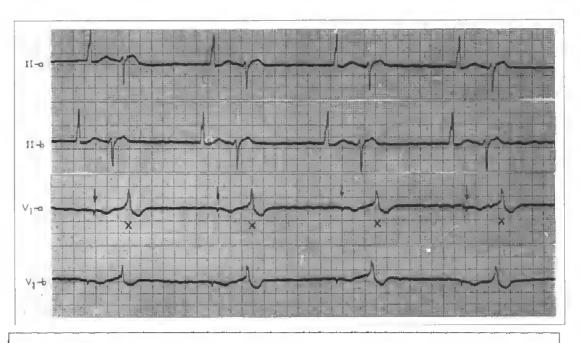




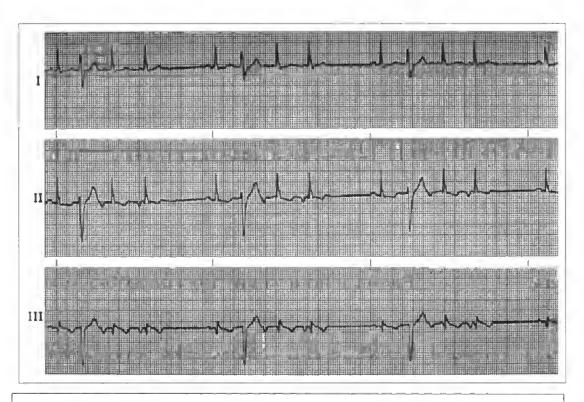




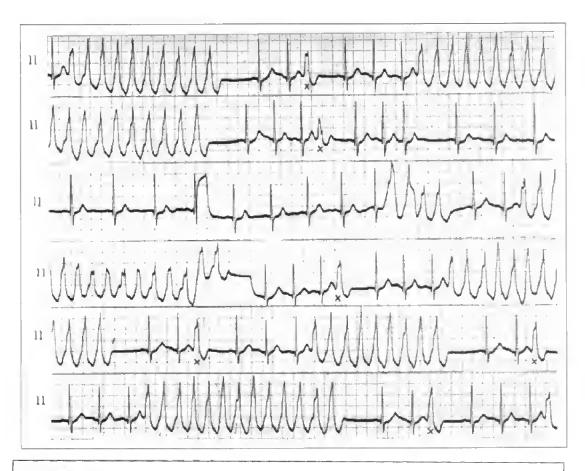




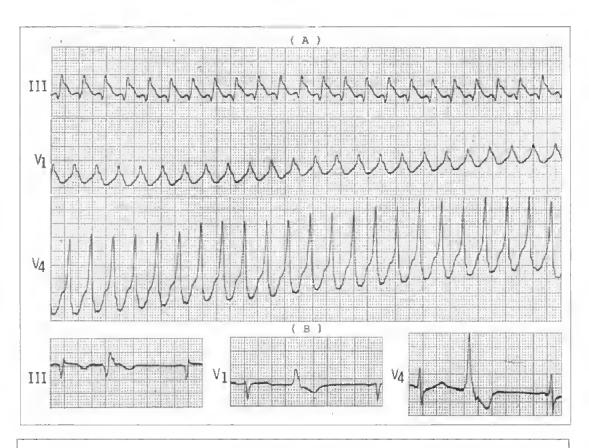




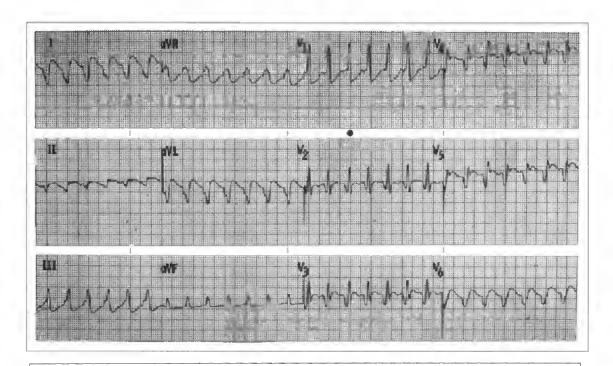




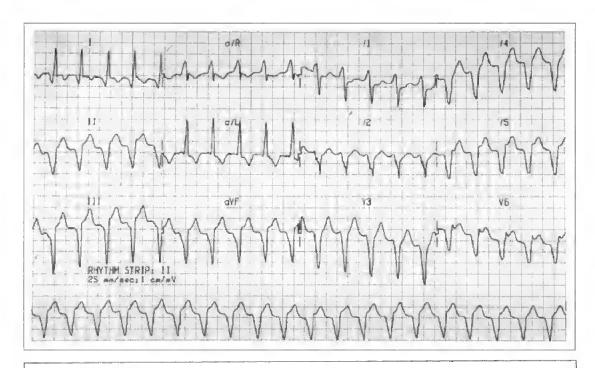




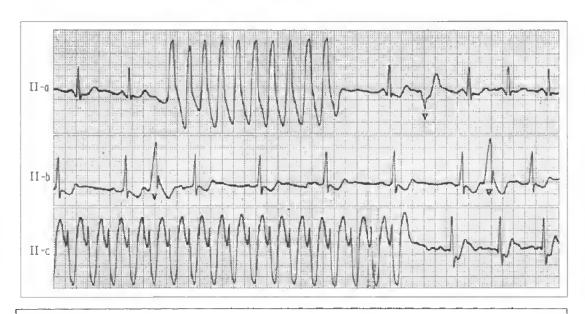




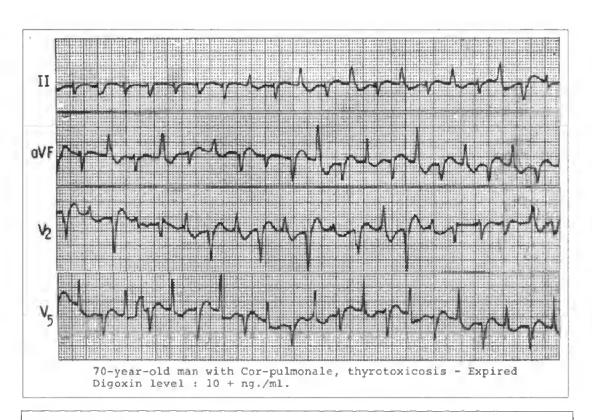




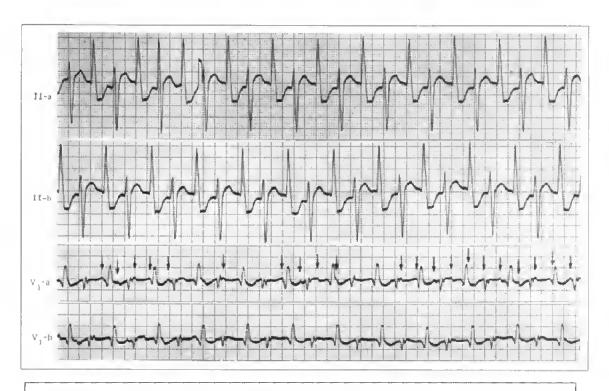








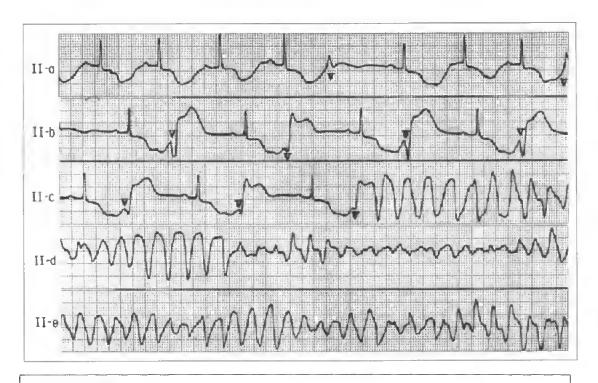




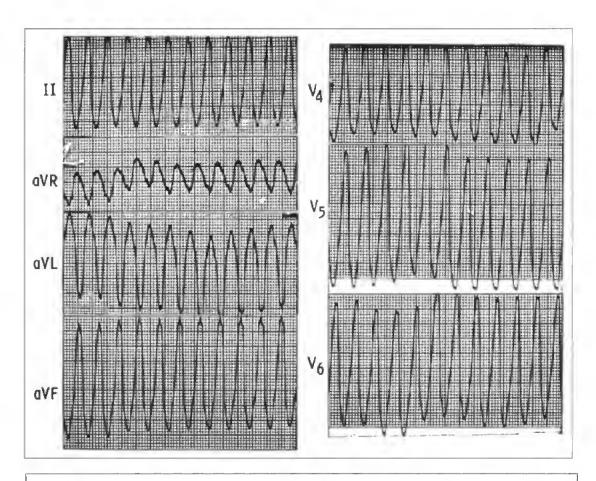






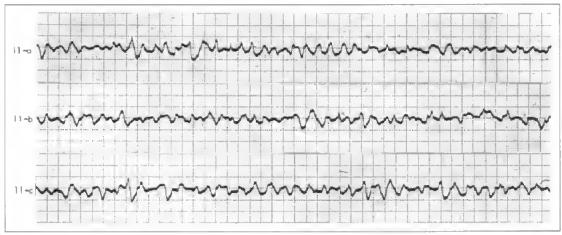




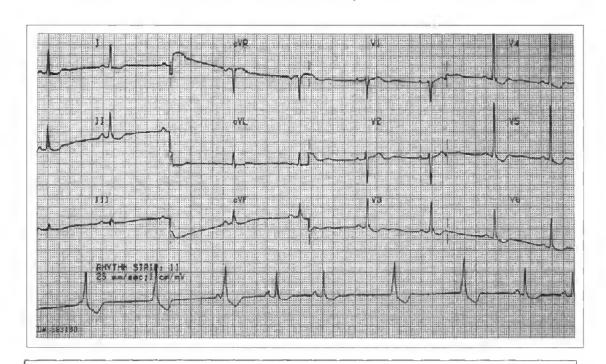


CASE 167

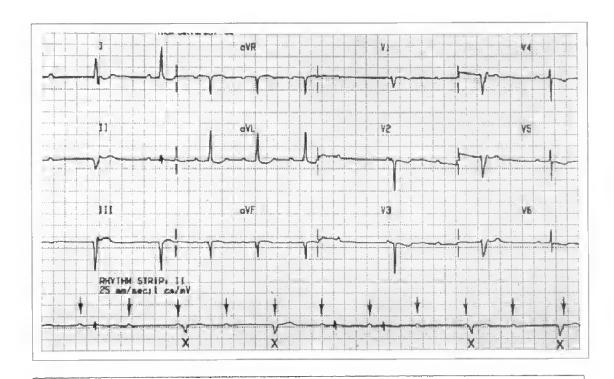




CASE 168
Your diagnosis:



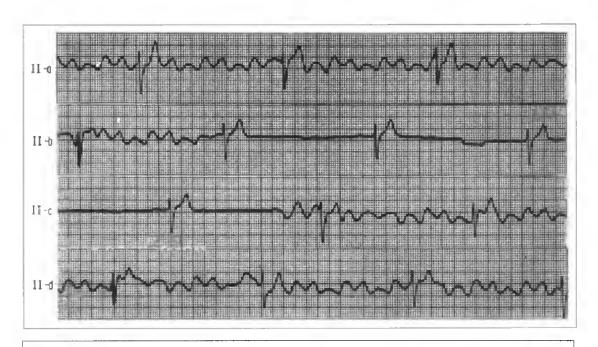






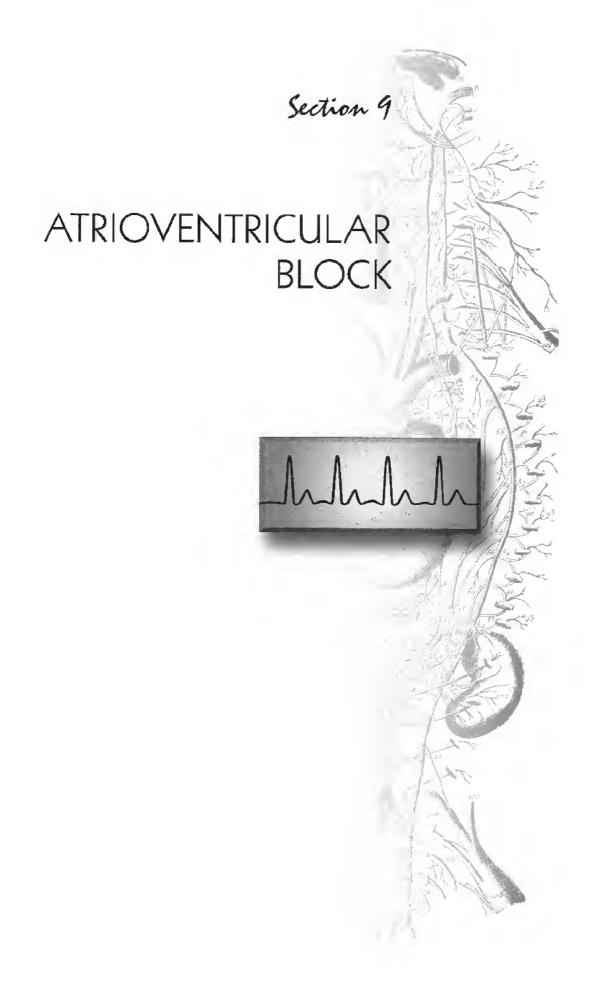


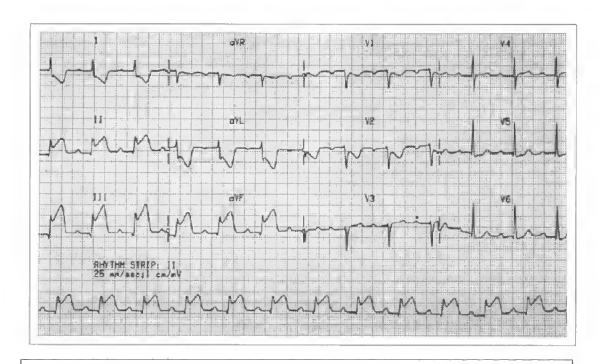




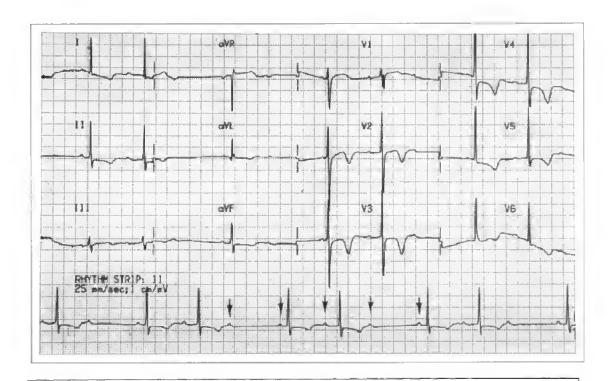


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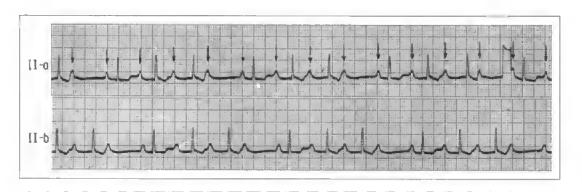




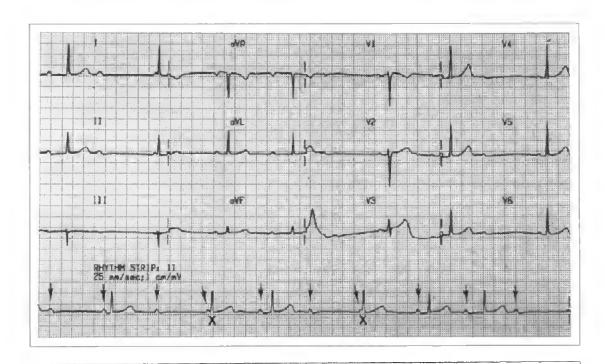




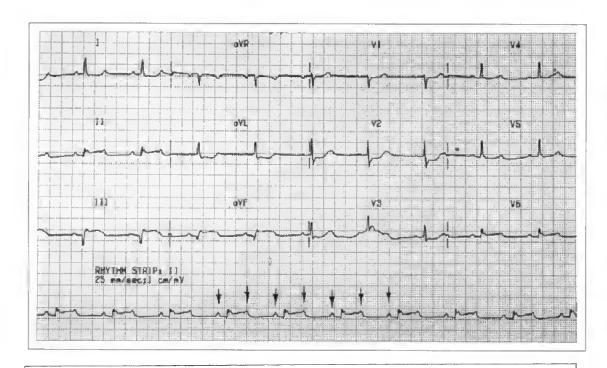




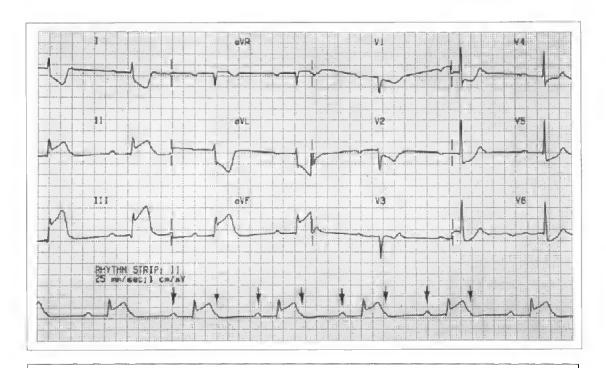




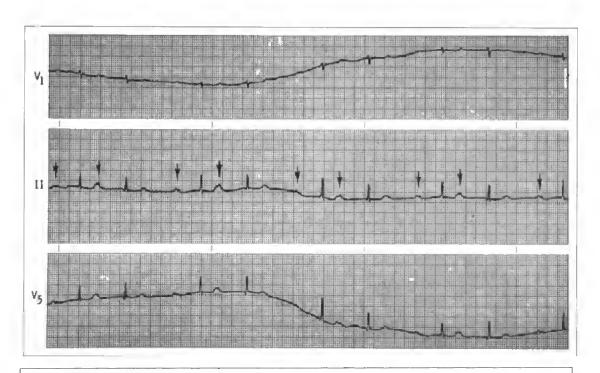




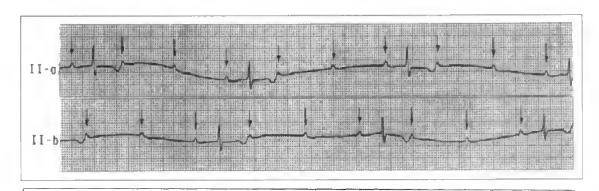




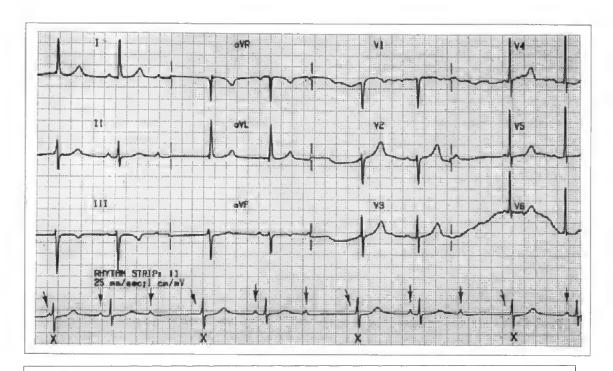




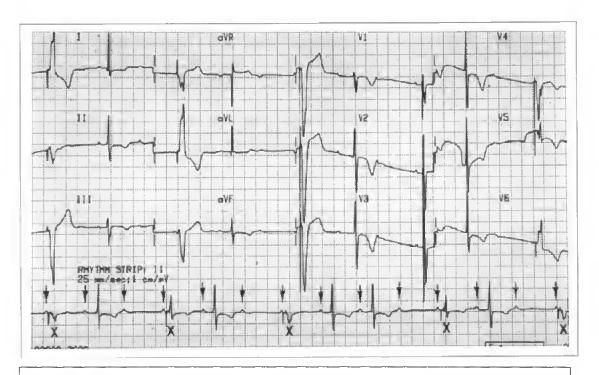




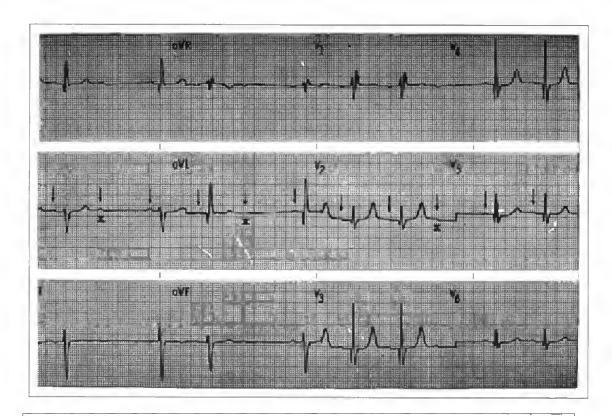




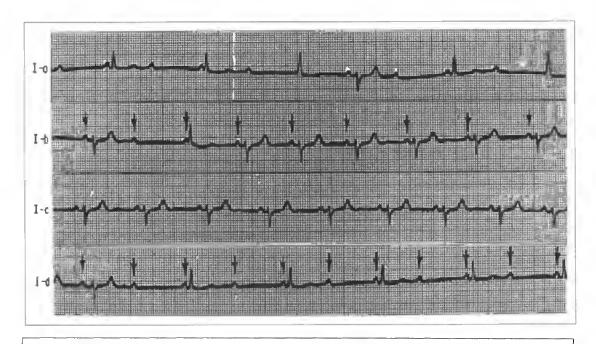




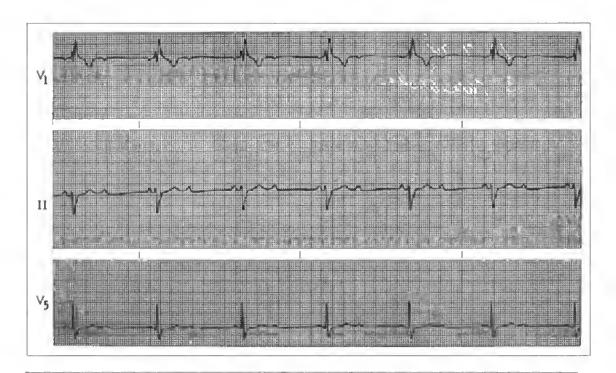




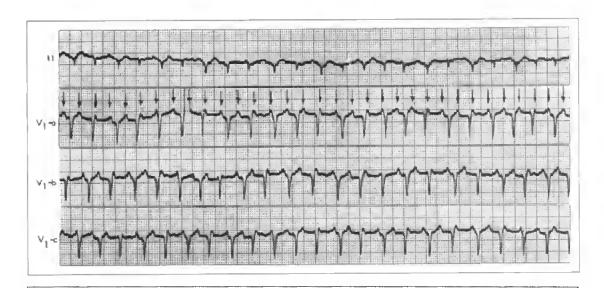








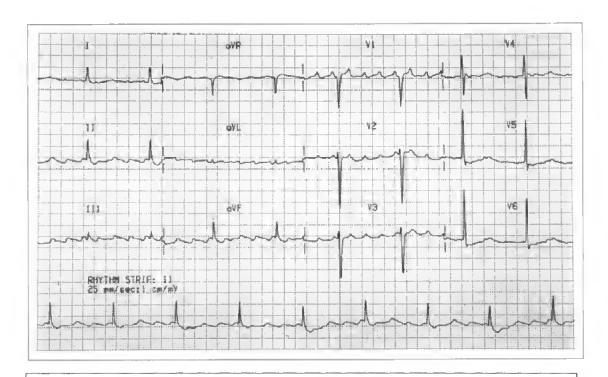




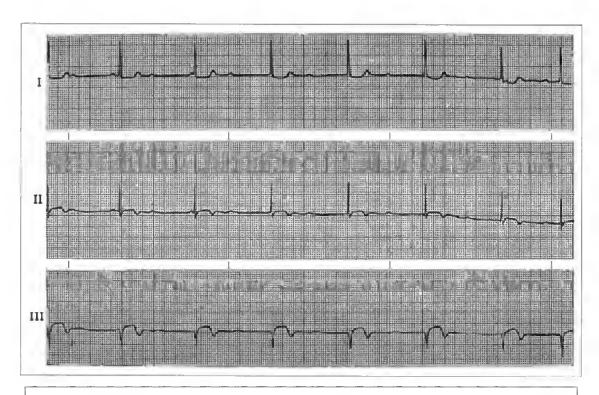




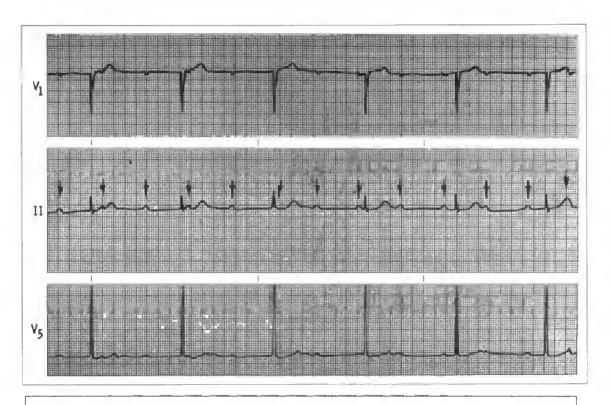




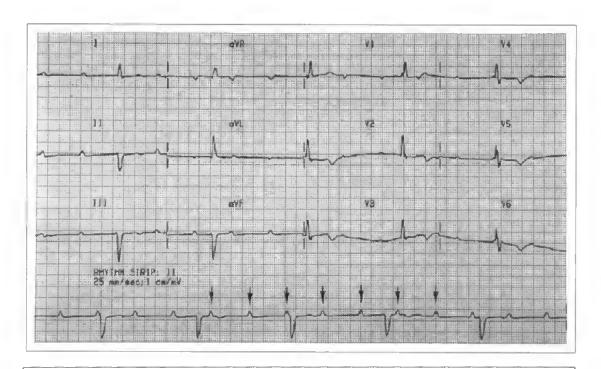










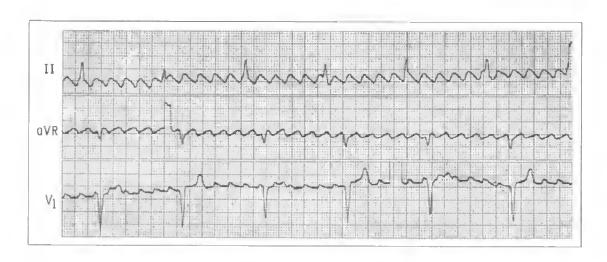






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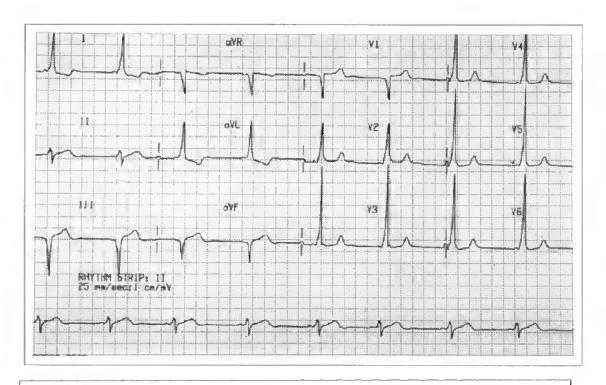




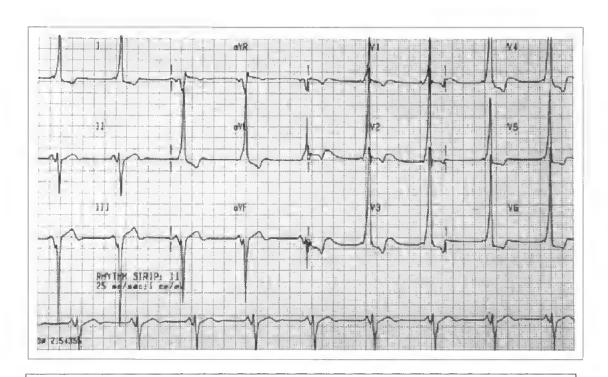


WOLFF-PARKINSON-WHITE SYNDROME (VENTRICULAR PREEXCITATION SYNDROME)

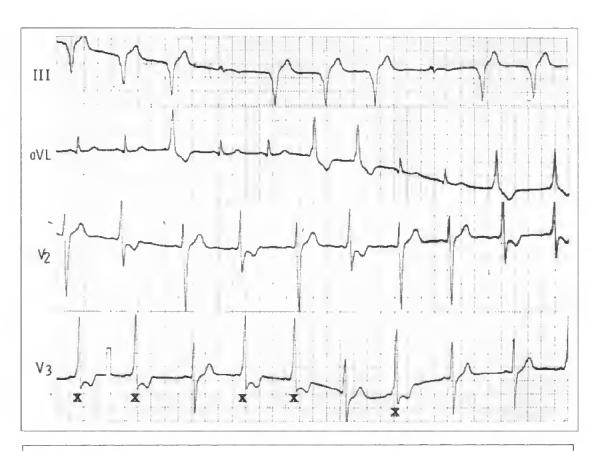


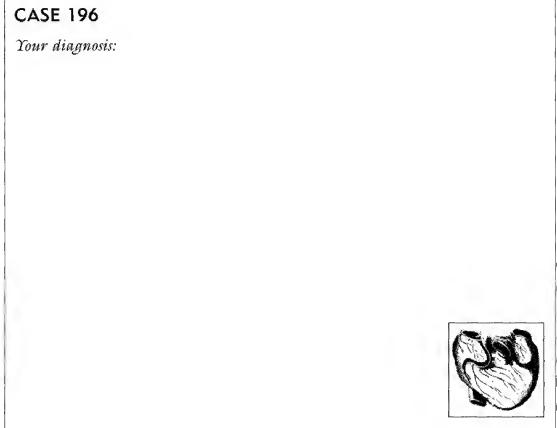


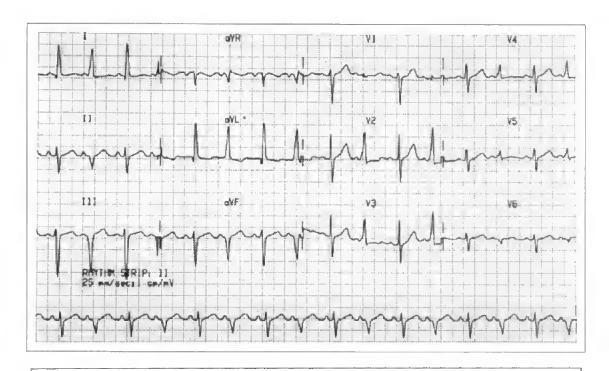




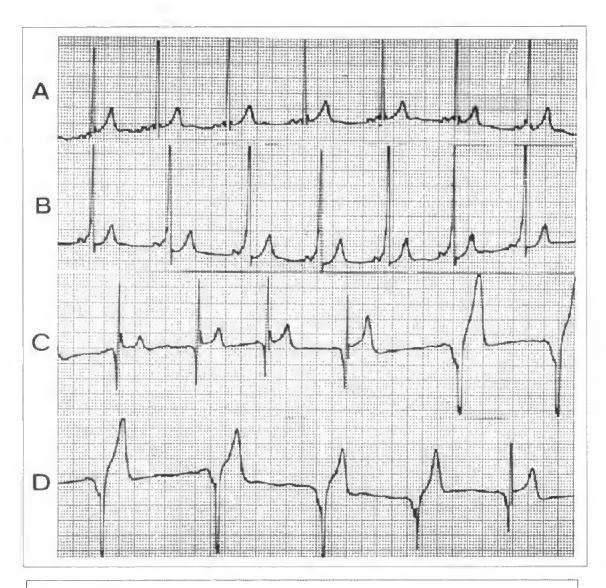




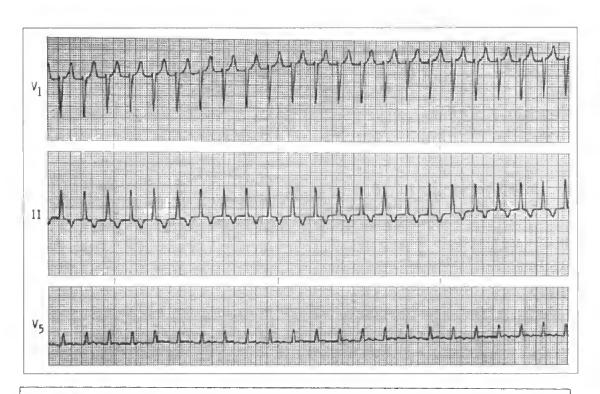




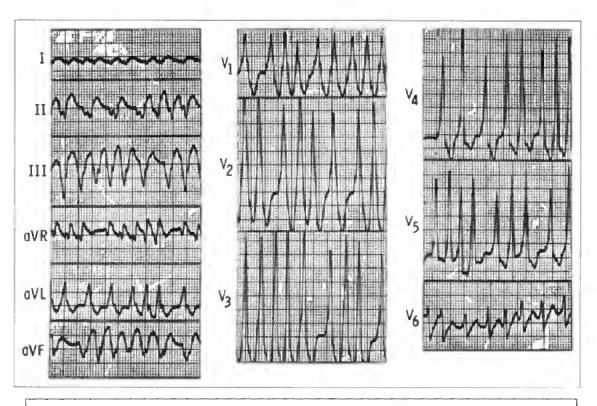




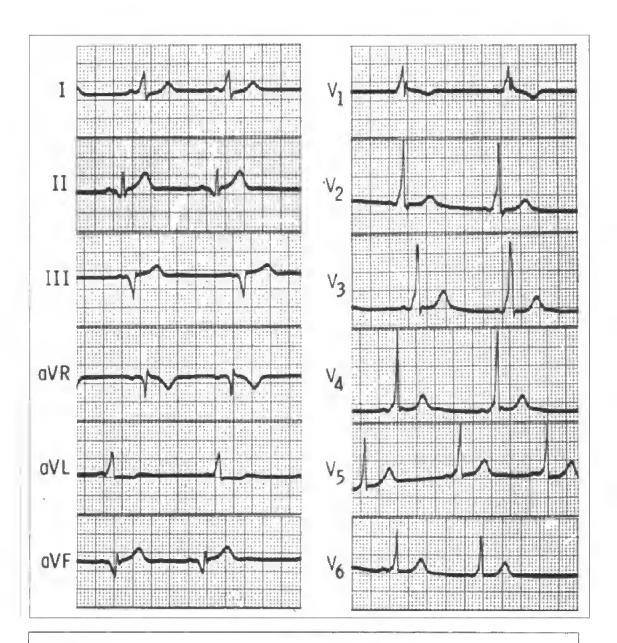




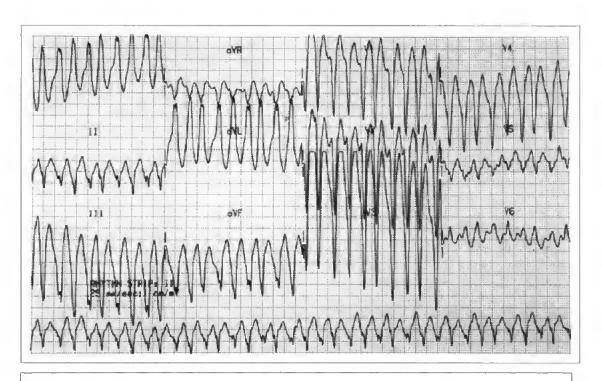




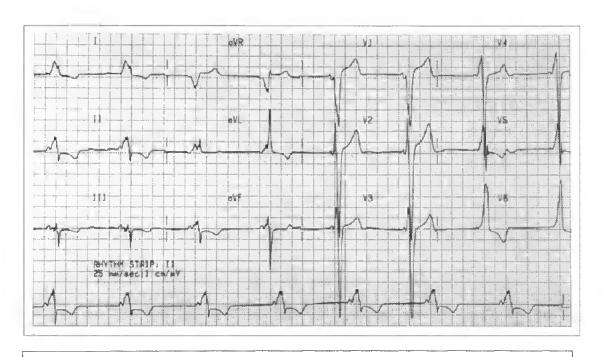




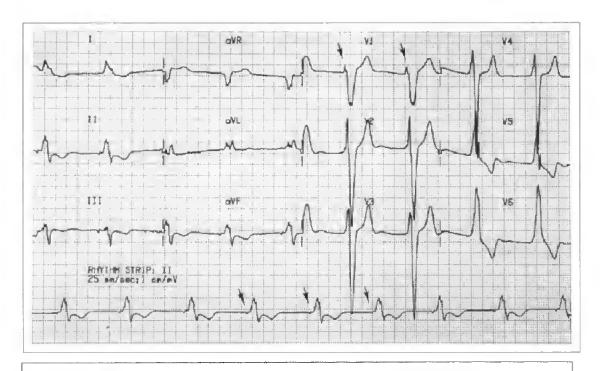




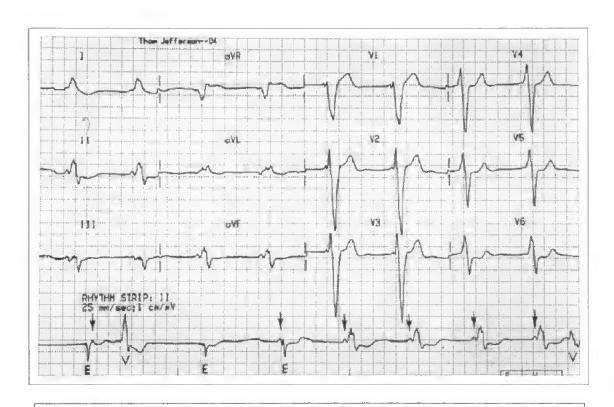




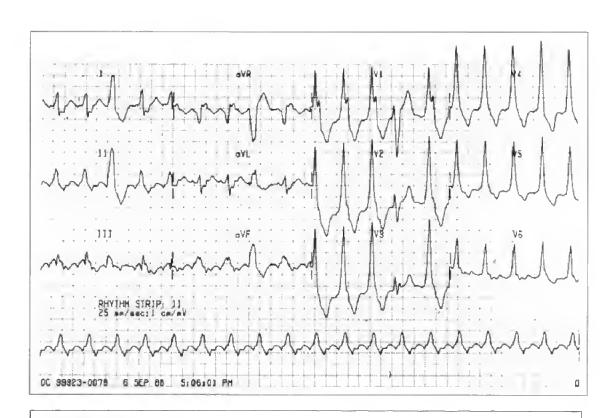




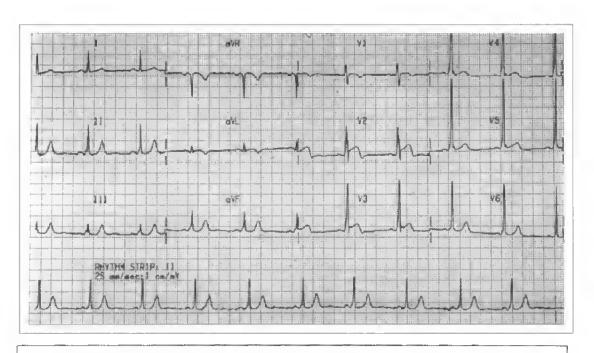






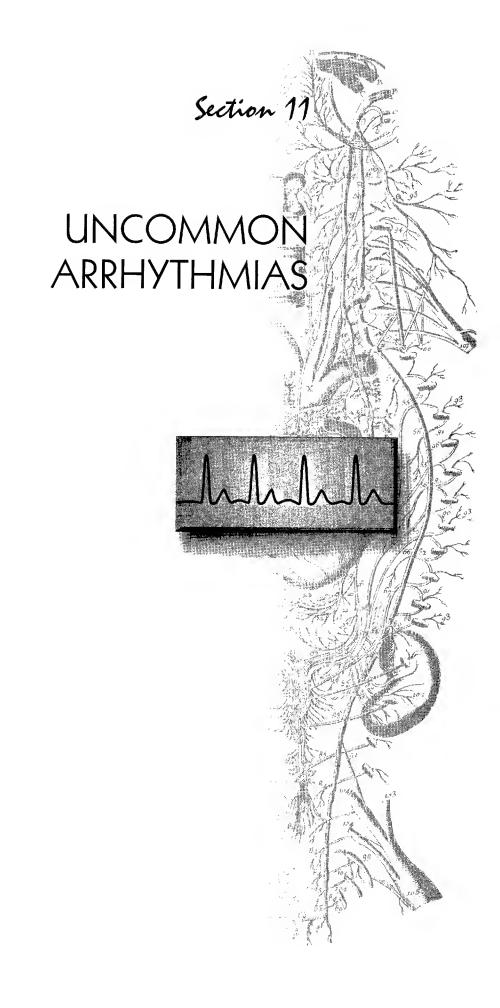


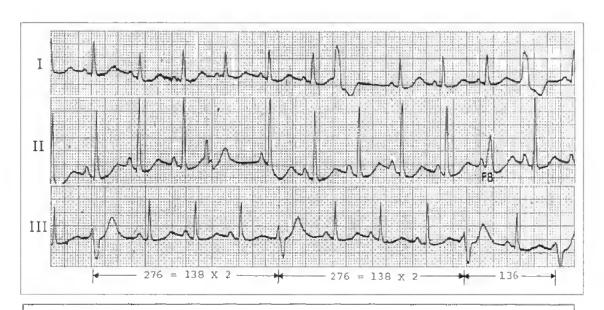




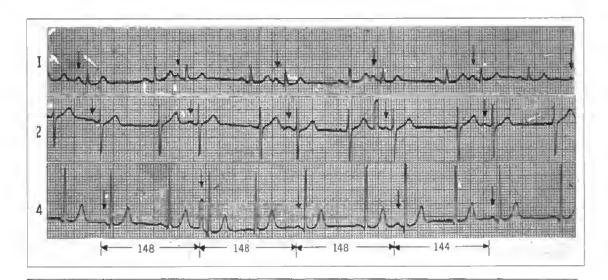


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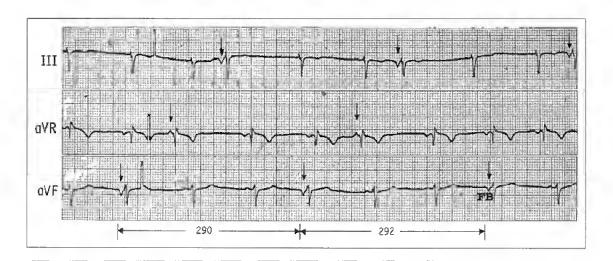




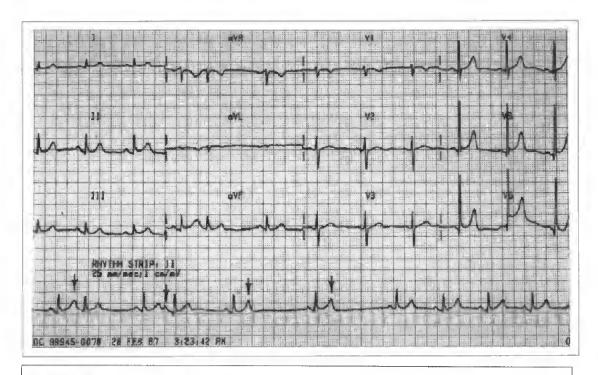




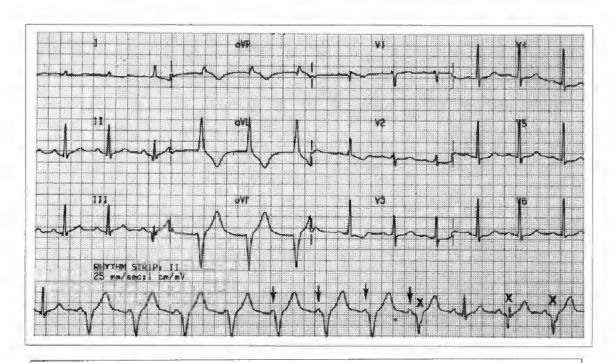




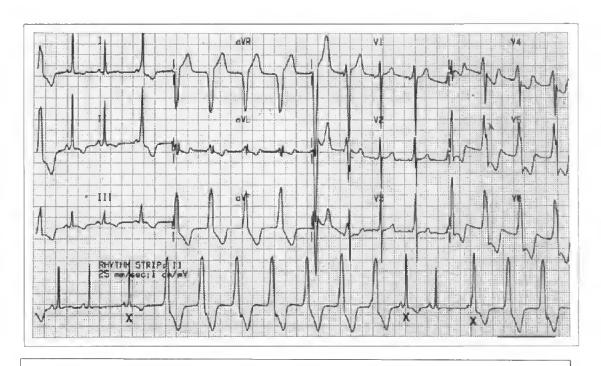




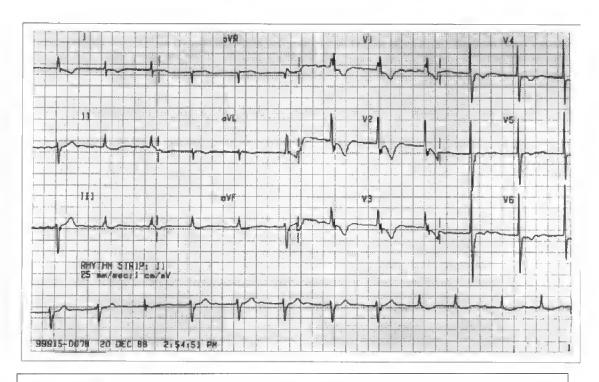




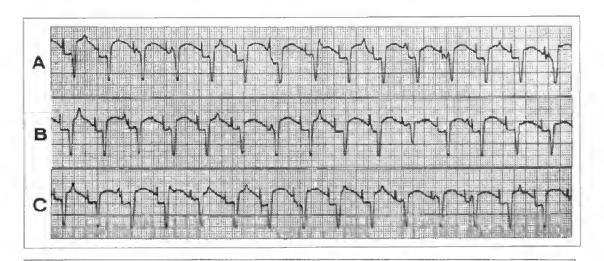




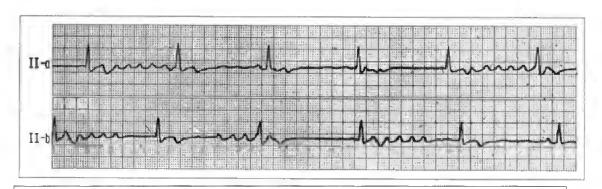




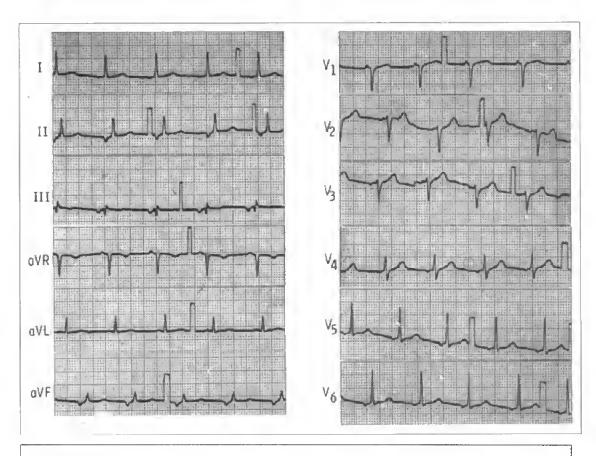




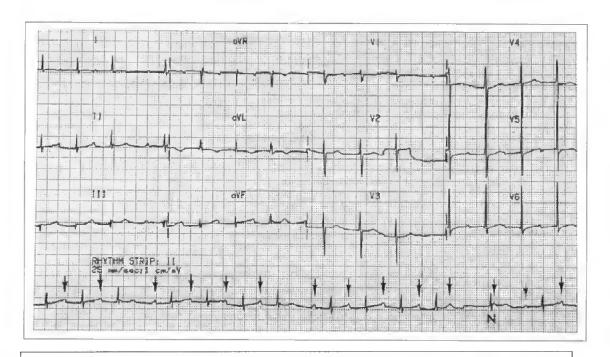




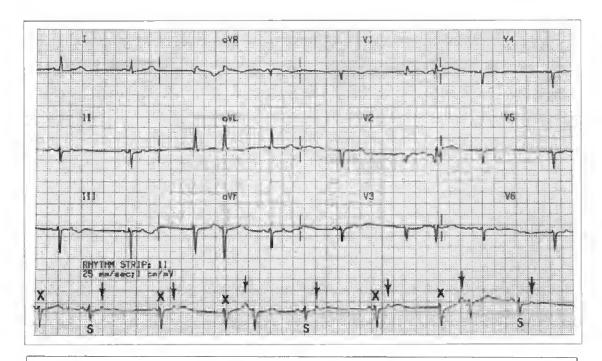




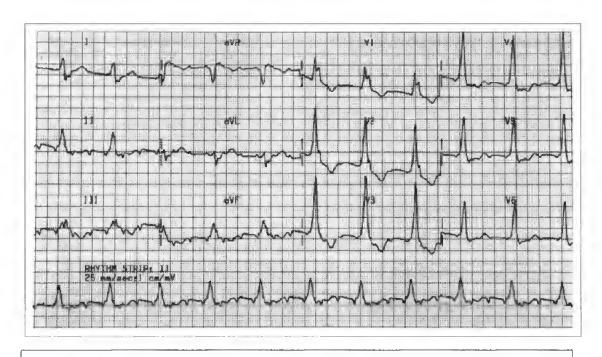




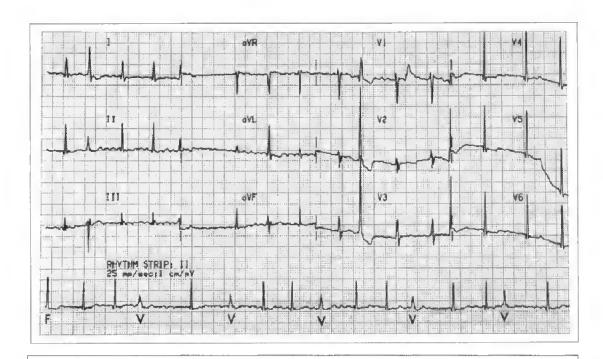




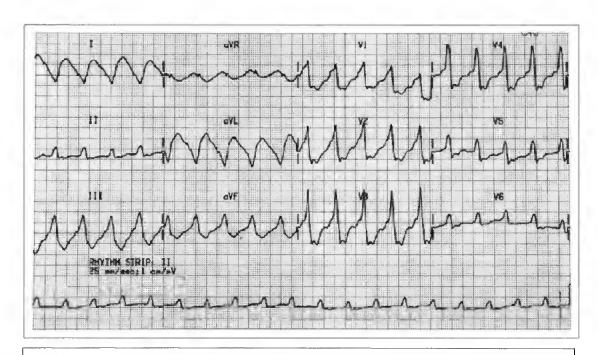




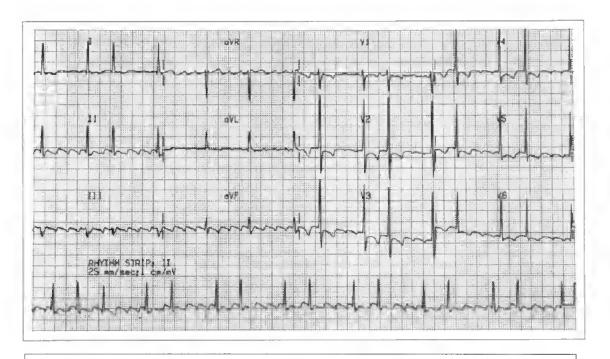




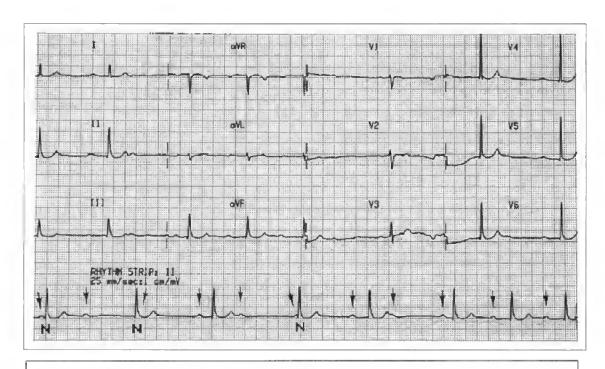




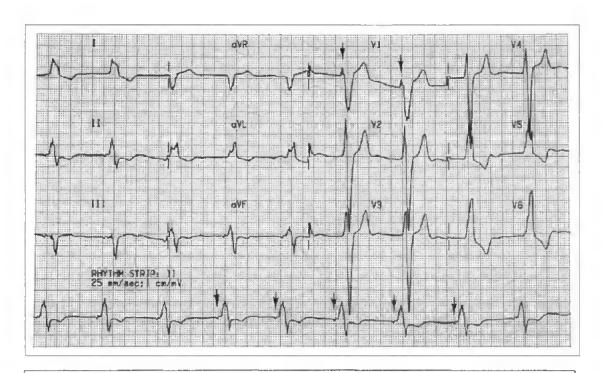




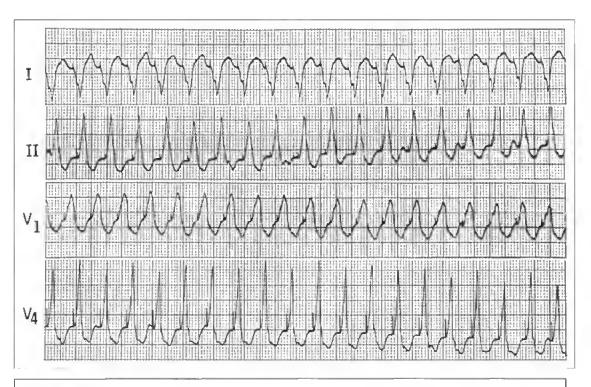








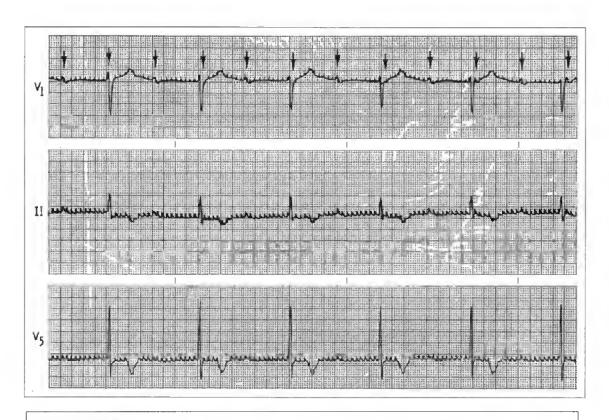




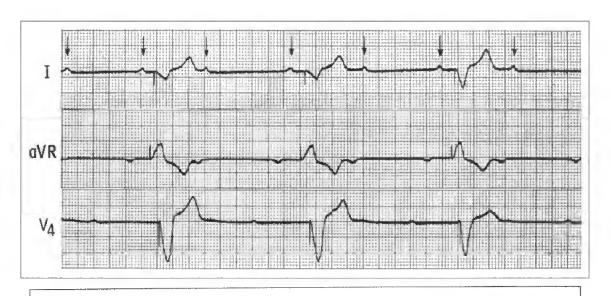




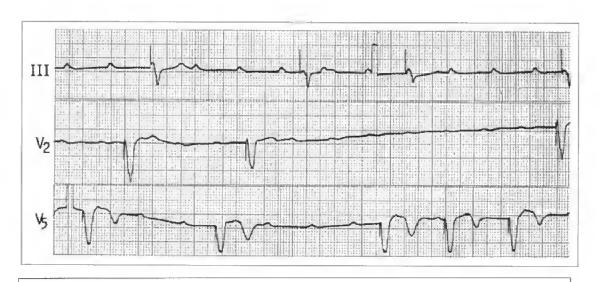




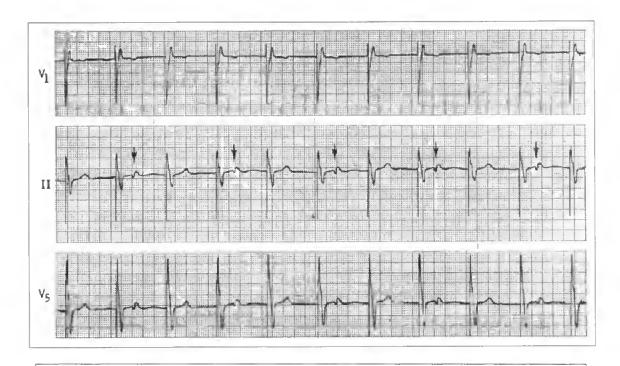




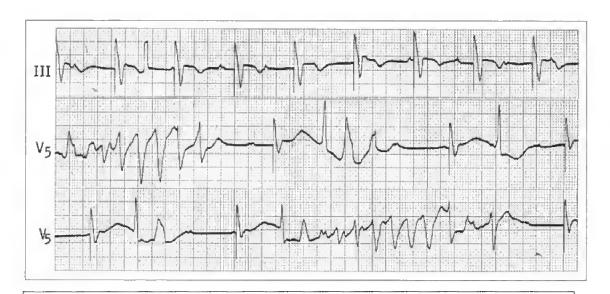




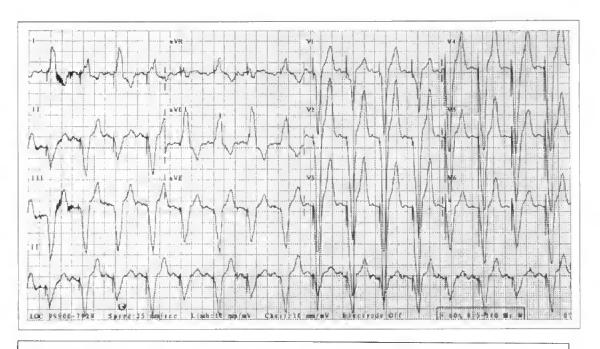






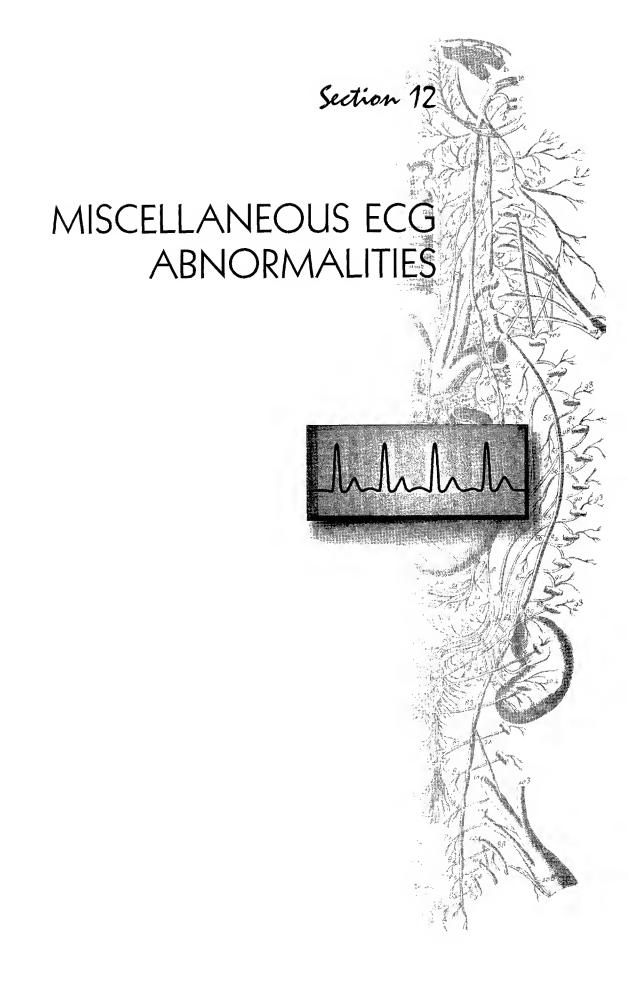


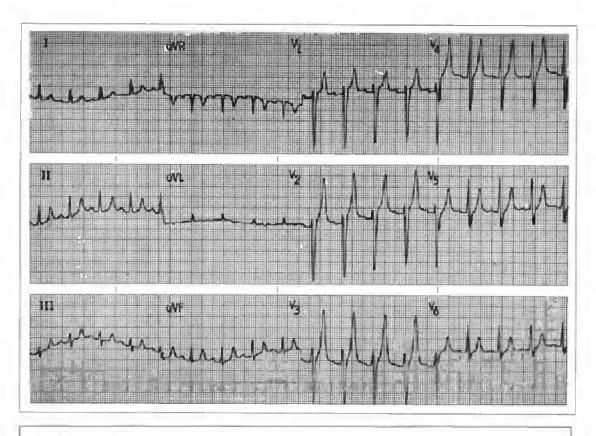




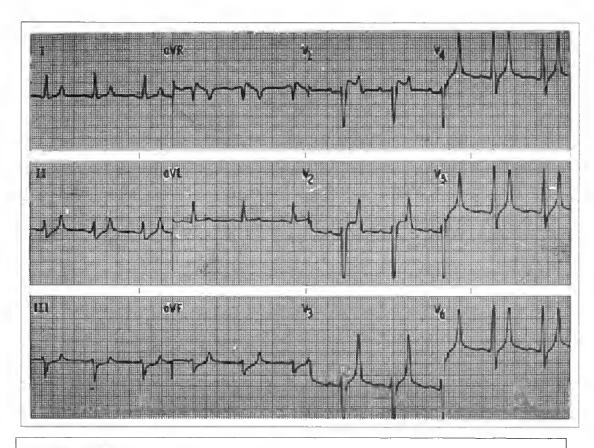


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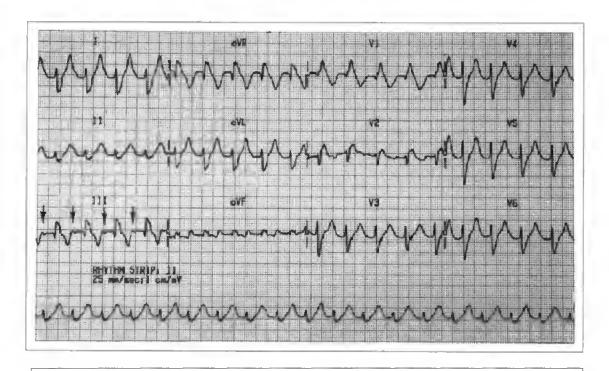








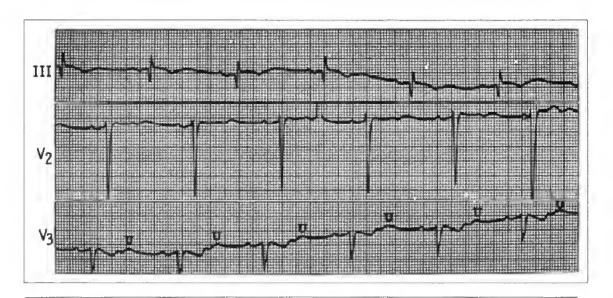




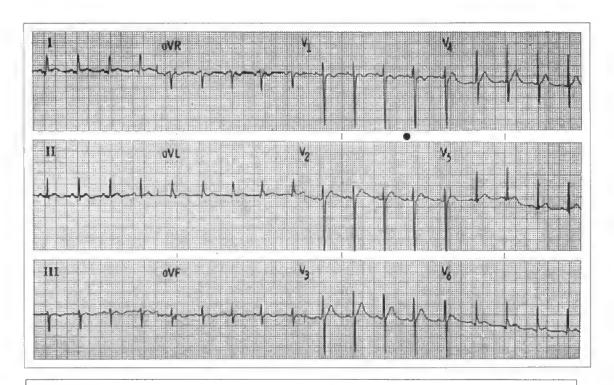




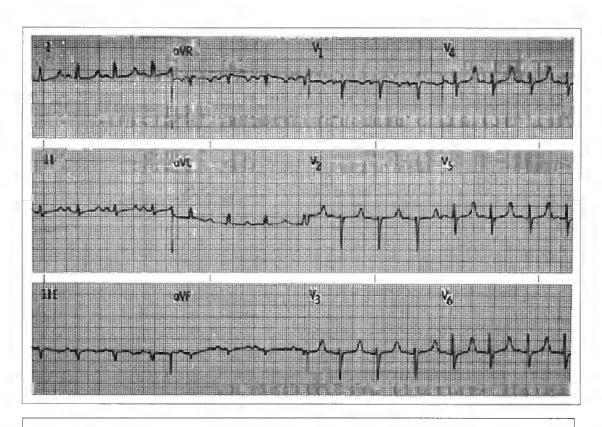




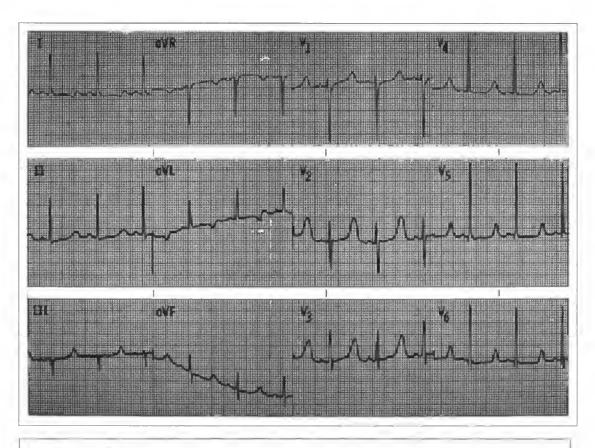




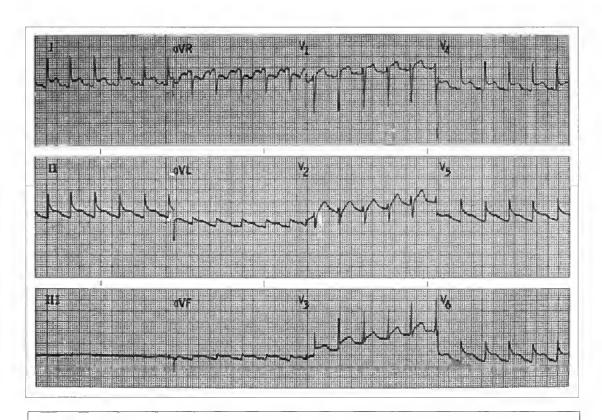




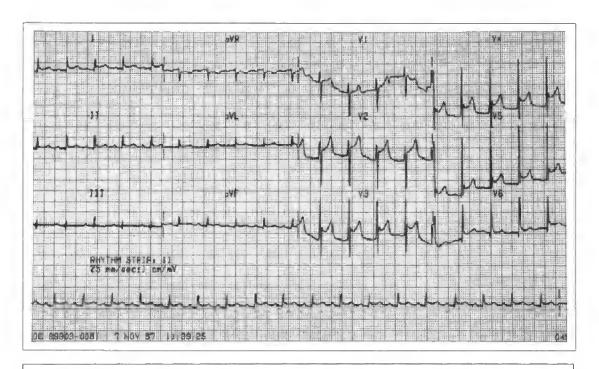




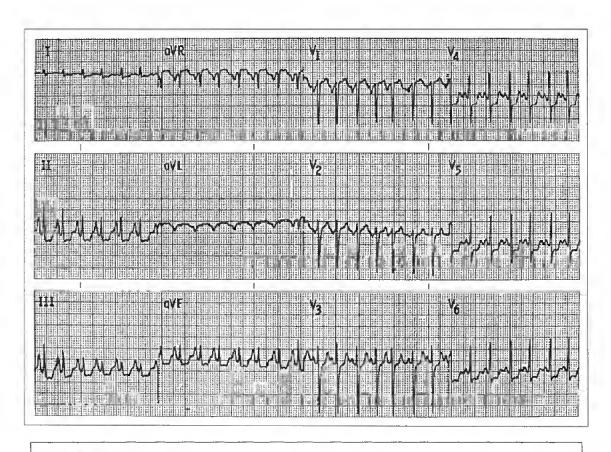




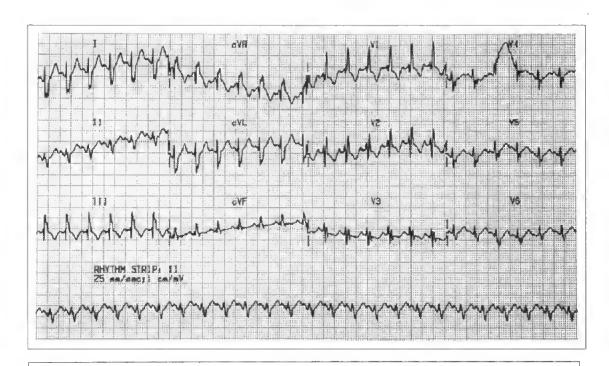




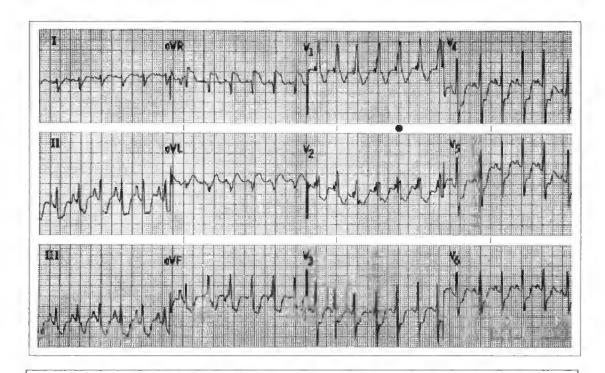




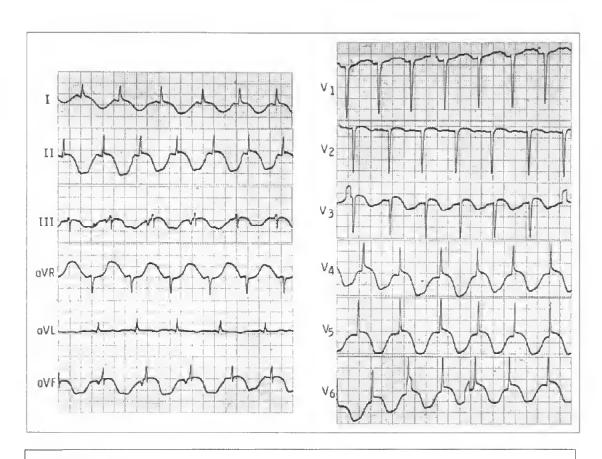




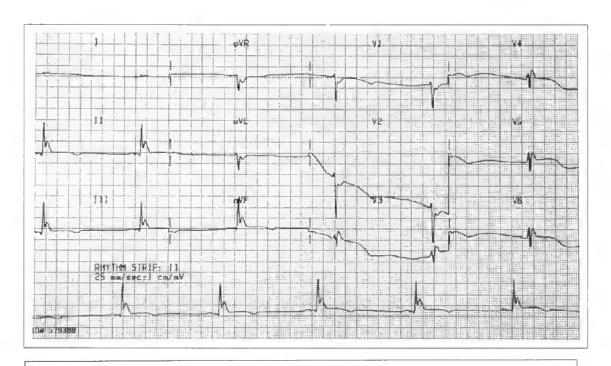




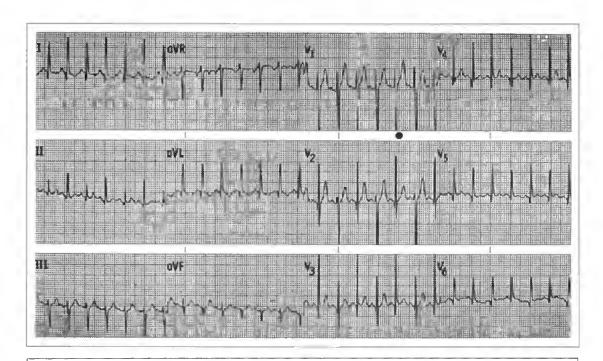








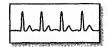




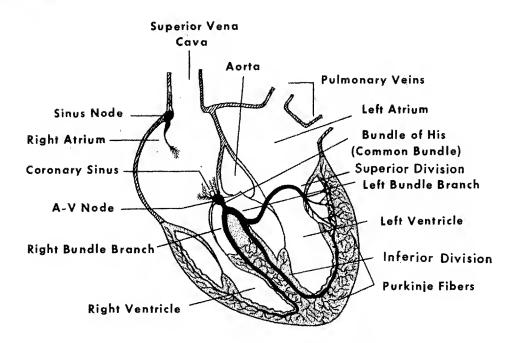








Case Descriptions



Section 1

Normal Electrocardiogram and Normal Variants

Case 1 Normal Electrocardiogram

In a normal electrocardiogram (ECG), the P wave is usually upright in lead II, whereas lead aVR reveals an inverted P wave. The P wave in lead V_1 exhibits a biphasic P wave. The average heart rates are between 60 and 75 beats/min.

FIVE DIAGNOSTIC CRITERIA FOR NORMAL SINUS RHYTHM (NSR)

- P wave of sinus origin (normal mean axis of P wave)
- Constant and normal PR interval (0.12–0.20 second)
- Constant P wave configuration in a given lead
- Rate between 60 and 100 beats/min
- Constant P-P (or R-R) interval (cycle)

Case 2 Sinus Arrhythmia with Wandering Atrial Pacemaker

The diagnosis of wandering atrial pacemaker (WAP) is made on the basis of varying configurations of the P waves. WAP is usually associated with sinus arrhythmia (see Chapter 5). Note that the P-P cycles vary considerably throughout the tracing. Sinus

arrhythmia with WAP is not uncommon among healthy young individuals.

Case 3 Juvenile T Wave Pattern and High Left Ventricular Voltage

This ECG tracing was obtained from a young healthy female. The cardiac rhythm is sinus arrhythmia, and it shows juvenile T wave pattern (JTWP) and high left ventricular voltage (HLVV). Both ECG findings are normal variants for young healthy people. Note the slightly inverted T waves in leads V_1 through V_3 with a biphasic T wave in lead V_4 (ITWP).

ECG FINDINGS IN NORMAL VARIANTS

- Juvenile T wave pattern (JTWP) in children and young adults
- Early repolarization pattern (ERP) in young black males
- Flat to biphasic T waves in many leads among athletes (athletic T wave change)
- High left ventricular voltage (HLVV) in children and young adults
- Short P-R interval
- Right axis deviation of the QRS complexes in children and young adults
- Sinus arrhythmia with or without wandering atrial pacemaker (WAP)

- Low voltage in obese individuals
- First-degree or Wenckebach atrioventricular (A-V) block in children and young adults (rare)

Case 4 Early Repolarization Pattern and High Left Ventricular Voltage

This ECG tracing reveals sinus bradycardia (rate: 56 beats/min) with ERP and HLVV in a healthy young black male. Note the J point elevation in leads V_3 through V_6 . Various normal variants were described earlier (see Case 3).

Case 5 Athletic T Wave Change (Normal Variant)

This ECG tracing was taken on a healthy young black athlete. It exhibits NSR and HLVV associated with flat to biphasic T waves in many leads (athletic T wave change—normal variant).

Section 2

Chamber Enlargement

Case 6 Left Atrial Enlargement with Right Ventricular Hypertrophy

This ECG tracing recorded from a patient with rheumatic mitral stenosis (MS) reveals left atrial enlargement (LAE) and right ventricular hypertrophy (RVH). The term, *P-mitrale* is used to designate LAE due to MS. These ECG abnormalities are the characteristic features of advanced MS.

DIAGNOSTIC CRITERIA OF LAE

- Broad (3 mm or more) and notched P waves in leads I, II, and aVL (also in leads III and aVF in some cases)
- A negative (inverted) component of P waves in leads V₁ through V₂, with a depth and width of I mm or more
- Coarse atrial fibrillation (AF); fibrillation waves in leads V₁ or V₂ of 1 mm or more

DIAGNOSTIC CRITERIA OF RVH

- Right axis deviation (RAD) of QRS complexes
- Tall (or relatively tall) R wave in lead V_I
- RR' wave—incomplete right bundle branch block (RBBB) pattern—in lead V₁
- Deep S waves in leads I, aVL, and V4 through V6
- Posterior axis deviation of QRS complexes with RAD (some cases)
- Secondary T wave change (strain pattern) in leads V₁ through V₃ (not every case)

The most reliable criteria include RAD with tall R wave in lead V_1 .

Case 7 Coarse Atrial Fibrillation due to Left Atrial Enlargement

These ECG rhythm strips, recorded from a patient with advanced MS, reveal coarse AF indicative of LAE (see Case 6). Note that the amplitude of AF waves is large (coarse AF).

Case 8 Right Atrial Enlargement (P-Congenitale) with Right Ventricular Hypertrophy

This ECG tracing taken on a child with severe congenital pulmonic stenosis (PS), reveals sinus rhythm with RAE as well as RVH. The term *P-congenitale* is used when RAE is due to various congenital cardiac anomalies. These ECG abnormalities are the characteristic features of severe PS.

DIAGNOSTIC CRITERIA OF RAE

- Tent-shaped and tall (3 mm or more) P waves in leads II, III, and aVF
- Less commonly, a positive (upright) component of P waves in leads V₁ through V₂, with an amplitude of 2 mm or more

Case 9 Right Atrial Enlargement (P-Pulmonale) and Right Ventricular Hypertrophy

This ECG tracing, obtained from an elderly woman with a far-advanced chronic obstructive pulmonary disease (COPD), demonstrates marked sinus tachycardia (rate: 152 beats/min) associated with RAE (P-pulmonale) as well as RVH. It is not uncommon to observe small q waves in leads V_1 through V_3 in severe RVH. A pseudo-diaphragmatic MI pattern is frequently observed in patients with advanced COPD. The diagnostic criteria of RAE and RVH were described earlier (see Cases 6 and 8).

Case 10 Pseudo P-Pulmonale due to Hypokalemia

This ECG tracing recorded from a patient with severe hypokalemia reveals marked sinus tachycardia (rate: 147 beats/min) associated with prominent U waves with peaked P waves (pseudo P-pulmonale). Inexperienced readers may not recognize prominent U waves, because the U waves are superimposed to the T waves. Peaked P waves (pseudo P-pulmonale) are not uncommon in advanced hypokalemia.

DIAGNOSTIC CRITERIA OF HYPOKALEMIA

• Prominent U waves (pronounced in leads V_3 through V_6) occur in many leads (the earliest ECG finding).

- Peaked P waves occur in leads II, III, and aVF (pseudo P-pulmonale) in advanced hypokalemia.
- Common arrhythmias are atrial premature contraction (APC), atrial tachycardia (AT) with or without atrioventricular (A-V) block, and supraventricular tachycardia (SVT).
- Less common arrhythmias are atrioventricular junctional tachycardia (A-V JT) or junctional escape rhythm (A-V JER).
- Ventricular premature contractions (VPCs) occur in far-advanced hypokalemia; ventricular tachycardia (VT) or fibrillation (VF) are rather uncommon.
- First-degree A-V block may occur; more advanced A-V block is rather uncommon.
- Hypokalemia predisposes patients to digitalisinduced arrhythmias.

The most common and the earliest ECG finding in hypokalemia is prominent U waves, especially in leads V_3 through V_6 . Pseudo P-pulmonale is less common in hypokalemia.

Case 11 Atrial Fibrillation with Left Ventricular Hypertrophy and Hypokalemia

This ECG tracing was recorded from an elderly woman with a long-standing hypertension associated with chronic congestive heart failure (CHF). The cardiac rhythm is AF with advanced A-V block. There are two ECG abnormalities that include LVH and prominent U waves indicative of hypokalemia. The prominent U waves are best shown in leads V_2 through V_5 .

DIAGNOSTIC CRITERIA OF LVH

- R wave in lead V₅ or V₆ of 26 mm or more
- R wave in lead V_5 or V_6 plus S wave in lead V_1 of 35 mm or more
- R wave in lead I of I5 mm or more
- R wave in lead I plus S wave in lead III of 25 mm or more
- R wave in lead aVL of 13 mm or more
- Secondary T wave change (strain pattern) in leads I, aVL, and V₄ through V₆

The most reliable diagnostic criteria of LVH are the secondary T wave change (strain pattern) with a tall R wave in lead V_5 or V_6 (whichever is taller) plus a deep S wave in lead V_1 . If there is no strain pattern, the ECG finding should be interpreted as "consider LVH by voltage criteria" for individuals older than 50 years of age. In younger people (50 years or younger), the term "high left ventricular voltage"

(HLVV) is used for this circumstance to signify that the ECG finding is a normal variant (see Cases 3 and 4).

Case 12 Left Ventricular Hypertrophy due to Aortic Stenosis and Possible Diaphragmatic Myocardial Infarction

This ECG tracing was obtained from a patient with aortic stenosis and coronary artery disease (CAD). The cardiac rhythm is sinus (rate: 90 beats/min), and marked LVH is present. In addition, the diagnosis of diaphragmatic MI is strongly considered (see Section 4). The diagnostic criteria of LVH were described earlier (see Case 11). The most common cause of LVH is hypertension, whereas aortic stenosis is the second most common underlying heart disease to cause LVH.

Case 13 Atrial Fibrillation Associated with Left Ventricular Hypertrophy, Intermittent Left Bundle Branch Block, and Anterior Myocardial Ischemia

These cardiac rhythm strips were obtained from an elderly woman with a long-standing CHF associated with hypertensive heart disease. The underlying cardiac rhythm is AF with advanced A-V block (ventricular rate: 56-115 beats/min). There are two kinds of QRS complexes because of intermittent LBBB (rate-dependent). Arrows indicate normally conducted beats. The diagnosis of LVH can be made during normal conduction (see QRS complexes with arrows). In addition, the T wave is inverted in lead V₂ due to anterior ischemia. In summary, these ECG rhythm strips reveal AF with advanced A-V block, LVH, intermittent LBBB (rate-dependent), and anterior ischemia (see Sections 3 and 4). The diagnostic criteria of LVH were described previously (see Case 11).

Case 14 Atrial Flutter and Right Ventricular Hypertrophy due to Rheumatic Heart Disease

This ECG tracing was taken on a young woman with known rheumatic heart disease (RHD). The cardiac rhythm is atrial flutter (indicated by arrows) with varying A-V response (predominately 2:1 A-V conduction). The diagnosis of RVH is established on the basis of incomplete RBBB pattern in leads V_1 through V_2 with RAD of the QRS complexes (QRS axis: $+120^{\circ}$). Note the flat T waves involving many leads (nonspecific T wave change). The diagnostic criteria of RVH were described previously (see Case 6).

Case 15 Right Ventricular Hypertrophy due to Pulmonic Stenosis

The ECG tracing, obtained from a young man with a congenital heart disease, demonstrates normal sinus rhythm (rate: 75 beats/min) and RVH. The diagnosis of RVH is entertained on the basis of RAD of the QRS complexes (QRS axis: +120°) with a tall R wave in lead V₁. In severe RVH, lead V₁ often shows a small q wave indicating that the ventricular septal activation is altered. This patient was found to have severe congenital pulmonic stenosis. The diagnostic criteria of RVH were described previously (see Case 6).

Case 16 Atrial Flutter-Fibrillation and Right Ventricular Hypertrophy due to Rheumatic Heart Disease

This ECG tracing was obtained from a patient with RHD. The underlying cardiac rhythm is atrial flutter-fibrillation (mixed form of atrial flutter and fibrillation) with advanced A-V block (ventricular rate: 62–70 beats/min). The diagnosis of RVH is established using the conventional criteria (see Case 6).

Case 17 Atrial Fibrillation with Left Atrial Enlargement and Biventricular Hypertrophy

This ECG tracing was taken on an elderly man with far-advanced CHF. The underlying cardiac rhythm is AF with advanced A-V block (ventricular rate: 70–85 beats/min). The diagnosis of biventricular hypertrophy (BVH) is established on the basis of the characteristic features of LVH in addition to RAD of the QRS complexes (QRS axis: +105°). In addition, LAE is strongly considered on the basis of coarse AF (see Case 6).

DIAGNOSTIC CRITERIA OF BVH

- LVH or LBBB in the precordial leads with RAD in the limb leads
- Tall (or relatively tall) R waves in all precordial leads with RAD in the limb leads (less reliable)
- Katz-Wachtel phenomenon: large amplitude of positive and negative components of the QRS complexes in leads V₂ through V₄ (usually RS complexes)
- P-pulmonale or P-congenitale in limb leads and LVH in the precordial leads (less reliable)

The most reliable diagnostic criteria of BVH are LVH in the precordial leads and RAD in the limb leads, as shown in this case.

Case 18 Biventricular Hypertrophy with Advanced Congestive Heart Failure due to Hypertensive Heart Disease

This ECG tracing, obtained from a middle-aged man with chronic CHF due to hypertensive heart disease, demonstrates sinus rhythm (rate: 94 beats/min) and BVH. The diagnosis of BVH is made on the basis of typical ECG findings of LVH on the precordial leads associated with a tall R wave in lead V_1 , RAD of the QRS complexes (QRS axis: $+105^{\circ}$), and large QRS amplitude in leads V_3 through V_5 (Katz-Watchel phenomenon) (see Case 17). Leads V_3 through V_6 are half-standardized.

Section 3

Intraventricular Block

Case 19 Right Bundle Branch Block

The underlying rhythm is sinus, and the diagnosis of right bundle branch block (RBBB) is made using the conventional criteria described as follows. Note that the QRS complexes are extremely broad throughout.

DIAGNOSTIC CRITERIA OF RBBB

- QRS duration of 0.12 second or more
- rSR' or an M pattern of QRS complex in lead V1
- \bullet Deep and slurred S waves in leads 1, aVL, and V_4 through V_6
- ullet Secondary ST segment and T wave change in leads V_1 through V_3

Case 20 Incomplete Right Bundle Branch Block

The underlying cardiac rhythm is sinus tachycardia, with a rate of 112 beats/min. The diagnosis of incomplete RBBB can be made using the conventional criteria (see Case 19). The diagnostic criteria of incomplete RBBB are essentially the same as those for RBBB except that the QRS duration is narrower than 0.12 second.

Case 21 Intermittent Right Bundle Branch Block, Rate-Independent

The cardiac rhythm is sinus, with a rate of 80 beats/min. It is obvious that RBBB occurs on every other beat (marked X) meaning intermittent RBBB, rate-independent. This ECG finding superficially simulates ventricular bigeminy (see Section 8).

Case 22 Intermittent Rate-Independent Right Bundle Branch Block with Left Ventricular Hypertrophy

These ECG rhythm strips reveal sinus rhythm (rate: 84 beats/min) with intermittent rate-independent

RBBB (marked X). Intermittent RBBB superficially mimics grouped ventricular premature contractions (VPCs) (see Section 8). In addition, the diagnosis of left ventricular hypertrophy (LVH) is made using the conventional criteria (see Case 11). Leads V_{6-a} and V_{6-b} are continuous.

Case 23 Incomplete Bifascicular Block Consisting of Left Anterior Hemiblock and Intermittent Bradycardia-Dependent Right Bundle Branch Block

The underlying cardiac rhythm is sinus (rate: 72 beats/min) with first-degree atrioventricular (A-V) block. There are frequent atrial premature contractions (APCs) producing atrial trigeminy (marked A). Arrows indicate sinus P waves. RBBB occurs intermittently following a post-ectopic pause—meaning intermittent bradycardia-dependent RBBB (marked X). Thus, a combination of fixed left anterior hemiblock (LAHB) and intermittent bradycardia-dependent RBBB indicates a partial (incomplete) bifascicular block (BFB). In addition, posterolateral myocardial ischemia must be considered, judging from the upright T wave in lead V₁ with inverted T wave in lead V₅ (see Section 4).

DIAGNOSTIC CRITERIA OF BILATERAL BUNDLE BRANCH BLOCK (BIFASCICULAR BLOCK AND TRIFASCICULAR BLOCK)

- RBBB with left anterior hemiblock (LAHB)
- RBBB with left posterior hemiblock (LPHB)
- Alternating LBBB and RBBB
- LBBB or RBBB with first- or second-degree A-V block (not every case)
- LBBB or RBBB with prolonged H-V interval (interval from the His potential to the ventricular deflection on the intracardiac ECG) more than 55 msec
- LBBB on one occasion and RBBB on another occasion
- Mobitz type II A-V block
- Any combination of the above findings
- Complete A-V block with ventricular escape (idioventricular) rhythm

Case 24 Atrial Fibrillation with Right Bundle Branch Block

These ECG rhythm strips exhibit AF with rapid ventricular response (ventricular rate: 170–180 beats/min) and RBBB. A grossly irregular ventricular cycle with no discernible P waves is the characteristic feature of AF (see Section 6).

Case 25 Intermittent Complete and Incomplete Right Bundle Branch Block with Diaphragmatic Myocardial Infarction

The cardiac rhythm is sinus bradycardia with a rate of 52 beats/min. It is interesting to note that complete and incomplete RBBB occur intermittently, and are not related to the heart rate change. Thus, this finding is a rate-independent phenomenon. In addition, the diagnosis of diaphragmatic myocardial infarction (MI) is established (see Section 4).

Case 26 Left Bundle Branch Block

This ECG shows sinus rhythm with LBBB. Leads V_1 through V_3 show a QS pattern that superficially resembles anteroseptal MI (pseudo-anteroseptal MI). This finding is due to LBBB.

DIAGNOSTIC CRITERIA OF LBBB

- QRS duration of 0.12 second or more
- \bullet Absence of septal q waves in leads I and V_4 through V_6
- rSR', an M pattern or broad R waves in leads I and V₄ through V₆
- Broad QS or rS waves in leads V1 through V3
- ullet Secondary ST segment and T wave change in leads I and V_4 through V_6

Case 27 Incomplete Left Bundle Branch Block

Sinus rhythm with incomplete LBBB (see Case 26). The QRS duration is relatively narrow.

Case 28 Intermittent (Rate-Dependent) Left Bundle Branch Block and a Ventricular Premature Contraction

Sinus arrhythmia with intermittent, rate-dependent LBBB and a VPC (the seventh beat). In this case, LBBB occurs when the heart rate increases. Thus, under this circumstance, intermittent LBBB is said to be tachycardia-dependent (see Case 26).

Case 29 Intermittent (Rate-Independent) Left Bundle Branch Block and Atrial Premature Contractions

Sinus rhythm with intermittent, rate-independent LBBB (see Case 26) and APCs (the sixth and seventh beats in the rhythm strip of lead V_6). In this case, LBBB occurs intermittently regardless of the heart rate change (rate-independent).

Case 30 Atrial Fibrillation with Left Bundle Branch Block

The cardiac rhythm is AF with very rapid ventricular response (ventricular rate: 160–185 beats/min). The QRS complexes are broad because of LBBB.

Ventricular tachycardia (VT) is superficially simulated, but a grossly irregular cardiac cycle excludes the possibility of VT (see Sections 6 and 8).

Case 31 Sinus Rhythm with 2:1 Atrioventricular Block Associated with Left Bundle Branch Block (Variant of Mobitz Type II Atrioventricular Block)

The underlying cardiac rhythm is sinus, but there is 2:1 A-V block (ventricular rate: 31 beats/min). Arrows indicate sinus P waves. The diagnosis of LBBB can be established using the conventional criteria (see Case 26). When LBBB or RBBB is associated with 2:1 A-V block, the block is considered to represent infranodal block. Thus, this ECG finding is a variant of Mobitz type II A-V block in most cases (see Section 9).

Case 32 Left Bundle Branch Block with Anterior Myocardial Ischemia

The cardiac rhythm is sinus with a rate of 70 beats/min. The two ECG abnormalities are LBBB and diffuse anterior ischemia. It should be noted that the T waves are deeply and symmetrically inverted in leads V₂ through V₆. This finding indicates that the primary T-wave change (ischemic T wave) replaced the secondary T-wave change in LBBB (see Case 26). Therefore, anterior myocardial ischemia is associated with LBBB in this patient. Clinically, acute anterior MI is strongly considered in this circumstance (see Section 4).

Case 33 Intermittent Left Bundle Branch Block Associated with Anterior Myocardial Ischemia, Left Ventricular Hypertrophy, Atrial Premature Contractions, and Ventricular Premature Contractions

Leads V_{6-a} to V_{6-b} are continuous. The underlying cardiac rhythm is sinus (rate: 60 beats/min), but there are VPCs (marked V) as well as APCs (see Sections 6 and 8). LBBB occurs intermittently, but is not related to the heart rate change (rate-independent). Other ECG abnormalities include LVH and anterior myocardial ischemia during normal conduction (marked X) (see Cases 11 and 26, and Section 4).

Case 34 Left Bundle Branch Block Associated with Right Ventricular Hypertrophy

The cardiac rhythm is sinus (rate: 88 beats/min), and the diagnosis of LBBB can be made using the conventional criteria (see Case 26). In addition, the diagnosis of right ventricular hypertrophy (RVH) is considered on the basis of right axis deviation

(RAD) of the QRS complexes (QRS axis: +120°) associated with LBBB (see Case 6).

Case 35 Left Bundle Branch Block Associated with Diaphragmatic Myocardial Ischemia

This ECG reveals sinus rhythm (rate: 67 beats/min) and LBBB associated with diaphragmatic (inferior) myocardial ischemia (see Case 26 and Section 4). Note deeply and symmetrically inverted T waves in leads II, III, and aVF, which are diagnostic of diaphragmatic myocardial ischemia. Clinically, acute diaphragmatic MI is strongly suggested (see Section 4).

Case 36 Left Anterior Hemiblock Associated with Extensive Anterior Myocardial Infarction and Left Ventricular Hypertrophy

The cardiac rhythm is sinus with a rate of 64 beats/min. There are three ECG abnormalities: left anterior hemiblock (LAHB) (QRS axis: -70°), acute extensive anterior MI, and LVH (see Section 4 and Case II).

DIAGNOSTIC CRITERIA OF LAHB

- Marked left axis deviation (LAD) (-45° to -90°) of QRS complexes
- A small q wave in lead I and a small r wave in lead III (not obvious in every case)
- Little or no prolongation of the QRS duration
- No evidence of other factors responsible for LAD

Case 37 Left Posterior Hemiblock Associated with Anteroseptal Myocardial Infarction and a Ventricular Premature Contraction

The underlying cardiac rhythm is sinus (rate: 97 beats/min), but there is a VPC in lead V_1 (the fourth beat). There are two ECG abnormalities: left posterior hemiblock (LPHB) (QRS axis: +I15°) and anteroseptal MI (see Section 4). In addition, left atrial enlargement is suggested (see Case 6).

DIAGNOSTIC CRITERIA OF LPHB

- Marked RAD (+105° to +180°) of the QRS complexes
- A small r wave in lead I and a small q wave in lead III (not obvious in every case)
- Little or no prolongation of the QRS duration
- No evidence of other factors responsible for RAD

Case 38 Intermittent Left Anterior Hemiblock, Rate-Dependent and Slow Atrial Tachycardia

The underlying cardiac rhythm is sinus (rate: 80 beats/min; the last four beats in the rhythm strip of lead II), but a slow atrial tachycardia (rate: I00 beats/min) occurs intermittently (see Section 6). Interestingly, LAHB (see Case 36) occurs only during the faster ventricular rate—meaning it is rate-dependent, and specifically tachycardia-dependent. In addition, LVH is suggested.

Case 39 Intermittent Complete and Incomplete Right Bundle Branch Block Associated with Recent Diaphragmatic Myocardial Infarction

A 50-year-old obese woman with various coronary risk factors was brought to the emergency room with acute chest pain of I to 2 hours in duration. The cardiac rhythm is sinus with a rate of 95 beats/min. With close observation, most readers should be able to recognize that there are two kinds of QRS complexes. This ECG represents intermittent, rate-independent complete and incomplete RBBB (see Case I9). Another ECG abnormality is recent diaphragmatic (inferior) MI (see Section 4). In addition, diffuse anterior myocardial ischemia is suggested.

Case 40 Bifascicular Block Consisting of Right Bundle Branch Block and Left Anterior Hemiblock

The cardiac rhythm is sinus (rate: 62 beats/min), and the ECG abnormality is bifascicular block (BFB), consisting of RBBB and LAHB (QRS axis: -65°) (see Cases 19, 23, and 36).

Case 41 Atrial Fibrillation Associated with Bifascicular Block, Consisting of Right Bundle Branch Block and Left Anterior Hemiblock

The cardiac rhythm is AF (see Section 6) with rapid ventricular response (ventricular rate: 125-165 beats/min). The diagnosis of BFB consisting of RBBB and LAHB (QRS axis estimated to be -60°) can be made using the conventional criteria (see Case 23).

Case 42 Bifascicular Block Consisting of Right Bundle Branch Block and Left Posterior Hemiblock and Possible Posterior Myocardial Infarction

The cardiac rhythm is sinus with a rate of 75 beats/min. The diagnosis of BFB, consisting of

RBBB and LPHB (the QRS axis: $+105^{\circ}$), can be readily made using the conventional criteria (see Case 23). In addition, the tall R wave in lead V_1 suggests posterior MI (see Section 4).

Case 43 Bifascicular Block Consisting of Right Bundle Branch Block and Left Posterior Hemiblock Associated with Anteroseptal Myocardial Infarction and Atrial Premature Contractions

This ECG tracing, obtained from a patient with known coronary artery disease (CAD), reveals BFB, consisting of RBBB and LPHB (the QRS axis: +120°) associated with old anteroseptal MI (see Case 23 and Section 4). In addition, old diaphragmatic MI is a remote possibility. Note the occasional APCs (e.g., the third beat in lead aVL, and the third beat in lead aVF).

Case 44 Incomplete Trifascicular Block Consisting of Right Bundle Branch Block and Left Anterior Hemiblock Associated with 2:1 Atrioventricular Block (Variant of Mobitz Type II Atrioventricular Block), Left Ventricular Hypertrophy and Probable Posterior Myocardial Infarction

The cardiac rhythm is sinus (atrial rate: 90 beats/min), with a 2:I A-V block that most likely represents Mobitz type-II A-V block (see Case 23 and Section 9). Arrows indicate sinus P waves. There are several ECG abnormalities, including BFB (consisting of RBBB and LAHB), LVH, and probable posterior MI. A combination of BFB and Mobitz type-II A-V indicates a partial trifascicular block (TFB) (see Case 23).

Case 45 Incomplete Trifascicular Block Manifested by Advanced Mobitz Type II Atrioventricular Block and Ventricular Escape Rhythm

The underlying cardiac rhythm is sinus (atrial rate: 72 beats/min), with an advanced A-V block that most likely represents a Mobitz type-II A-V block (see Section 9). Arrows indicate sinus P waves. There are frequent ventricular escape beats (VEBs, marked E), meaning that the block is most likely distal to the A-V node (infranodal block). Other ECG abnormalities include RBBB and lateral myocardial ischemia. A combination of advanced Mobitz type-II A-V block, RBBB, and frequent ventricular escape beats is diagnostic of a partial but advanced TFB (see Case 23). In addition, a possi-

bility of anteroseptal MI is considered (the pathologic Q wave in lead V_1).

Case 46 Partial Advanced Trifascicular Block Manifested by Advanced Mobitz Type II Atrioventricular Block and Right Bundle Branch Block with Ventricular Escape Rhythm

The underlying cardiac rhythm is sinus (atrial rate: 76 beats/min), but complete A-V block occurs intermittently leading to intermittent ventricular escape rhythm (VER, marked X). Note frequent ventricular fusion beats (marked FB). Arrows indicate sinus P waves. Another ECG abnormality is RBBB. A combination of advanced Mobitz type-II A-V block (intermittent complete A-V block), RBBB, and intermittent VER is diagnostic of a partial but advanced TFB (see Case 23).

Case 47 Partial Trifascicular Block Manifested by First-Degree Atrioventricular Block, Right Bundle Branch Block, and Intermittent Left Anterior Hemiblock

The cardiac rhythm is sinus (rate: 74 beats/min) with first-degree A-V block (PR interval: 0.24 second). The ECG abnormalities include RBBB and intermittent LAHB leading to a partial BFB. A combination of the above-mentioned ECG findings is suggestive of a partial TFB (see Case 23).

Case 48 Diffuse (Nonspecific) Intraventricular Block due to Quinidine Toxicity

This ECG tracing demonstrates sinus rhythm (rate: 96 beats/min) with diffuse (nonspecific) intraventricular block as a result of quinidine toxicity. LBBB is superficially simulated, but the presence of septal q waves in leads V_5 through V_6 is against the diagnosis of LBBB (see Case 26).

VARIOUS CONDITIONS THAT CAUSE DIFFUSE (NONSPECIFIC) INTRAVENTRICULAR BLOCK

- *Drugs:* Quinidine, procainamide, and other similar antiarrhythmic agents
- Electrolyte Imbalance: Hyperkalemia
- Heart Disease: Cardiomyopathies, MI, and other advanced heart diseases
- After Cardiac Arrest: Conditions of cardiac or non-cardiac origin
- Miscellaneous: Hypothermia and advanced age

Case 49 Diffuse (Nonspecific) Intraventricular Block due to Severe Hyperkalemia Associated with Electrical Alternans

This ECG tracing was obtained from a newborn infant with advanced renal failure as a result of irre-

versible kidney malformation. Severe hyperkalemia is manifested by flat P waves and diffuse intraventricular block. In addition, there is electrical alternans, involving ST segment and T waves (repolarization electrical alternans). Electrical alternans is observed in this infant as a result of uremic pericarditis (see Section 12).

ECG FINDINGS IN HYPERKALEMIA

- Tent-shaped and tall T waves with narrow base
- Flat P waves
- A-V block (varying degrees)
- Intraventricular block (various types)
- Ventricular arrhythmias (advanced cases)
- Ventricular standstill (far-advanced cases)

Case 50 First-Degree Atrioventricular Block and Diffuse (Nonspecific) Intraventricular Block due to Hyperkalemia Associated with Hypocalcemia

This ECG tracing was recorded from a patient with advanced chronic renal failure. The cardiac rhythm is sinus arrhythmia (rate: 54–80 beats/min) with first-degree A-V block. Hyperkalemia is manifested by diffuse intraventricular block, peaked T waves, and first-degree A-V block (see Case 49). In addition, a markedly prolonged QT interval secondary due to lengthening of the ST segment is observed as a result of coexisting hypocalcemia. It has been shown that hyperkalemia and hypocalcemia commonly coexist in patients with advanced renal failure.

ECG FINDINGS IN HYPOCALCEMIA

- Most common finding: Prolongation of the QT interval due to lengthening of the ST segment
- Less common finding: Flattening or inversion of the T waves
- Rare finding: Premature beats of various origins
- Common coexisting finding: Hyperkalemia

Case 51 Diffuse (Nonspecific) Intraventricular Block with Atrial Flutter-Fibrillation due to Hypothermia

This ECG tracing was obtained from a patient who had been in an auto accident on a very cold day. The cardiac rhythm is atrial flutter-fibrillation with advanced A-V block (ventricular rate: 35–56 beats/min). Hypothermia is manifested by diffuse intraventricular block, J-point elevation of the ST segment with prominent notching of the terminal portion of the QRS complex (termed the "Osborn wave"), atrial flutter-fibrillation, and grouped VPCs.

ECG FINDINGS IN HYPOTHERMIA

- Finding: Diffuse (nonspecific) intraventricular block
- Osborn wave: J-point elevation of the ST segment with prominent notching of the terminal portion of the QRS complex
- Cardiac arrhythmias: Sinus arrest, atrial flutter or fibrillation, VPCs, and A-V block

Case 52 Diffuse (Nonspecific) Intraventricular Block and Left Anterior Hemiblock due to Cardiomyopathy

This ECG tracing, obtained from a patient with idiopathic cardiomyopathy, reveals a sinus rhythm (rate: 75 beats/min) and LAHB (QRS axis: -60°) associated with diffuse intraventricular block (see Cases 36 and 48). Posterior and high lateral MI are a remote possibility (see Section 4).

Case 53 Diffuse (Nonspecific) Intraventricular Block due to Extensive Anterior Myocardial Infarction

This ECG tracing was recorded from a patient with acute heart attack. The cardiac rhythm is sinus tachycardia with a rate of 108 beats/min. The ECG abnormalities include acute extensive anterior MI associated with diffuse intraventricular block (see Case 48 and Section 4). In addition, diaphragmatic MI is a remote possibility.

Section 4

Myocardial Ischemia, Injury, and Infarction

Case 54 Diffuse Subendocardial Ischemia

This ECG tracing was obtained from a patient with acute chest pain. The cardiac rhythm is sinus with a rate of 68 beats/min. Diffuse subendocardial ischemia is manifested by large and broad upright T waves involving many ECG leads.

ECG FINDINGS IN SUBENDOCARDIAL ISCHEMIA

- Large and broad upright T waves (may involve many leads)
- Prolongation of the QT interval due to broad and large T wave
- No ST segment change
- No pathologic Q wave

Case 55 Diffuse Myocardial (Subepicardial) Ischemia

This ECG tracing was taken on a patient who was brought to the emergency room because of severe chest pain. The cardiac rhythm is sinus with a rate of 95 beats/min. The T waves are deeply and symmetrically inverted in practically every ECG lead. Thus, the diagnosis of diffuse myocardial ischemia is readily made. Non-Q wave myocardial infarction (MI) should be strongly considered when this ECG abnormality persists and the clinical picture of acute MI is also present.

ECG FINDINGS IN MYOCARDIAL (SUBEPICARDIAL) ISCHEMIA

- Deeply and symmetrically inverted T waves (may involve many leads)
- No ST segment change
- No pathologic Q wave

Case 56 Diffuse Myocardial Ischemia Associated with Intermittent Atrioventricular Junctional Escape Rhythm and Incomplete Atrioventricular Dissociation

This ECG tracing was taken on a patient with known coronary artery disease (CAD). Arrows indicate P waves. The cardiac rhythm is sinus bradycardia (atrial rate: 52 beats/min) with intermittent atrioventricular junctional escape rhythm (A-V JER) (ventricular rate: 56 beats/min) producing incomplete A-V dissociation (see Section 7). The T waves are inverted in many leads indicating diffuse myocardial ischemia (see Case 55).

Case 57 Incomplete Right Bundle Branch Block Associated with Posterior Myocardial Ischemia and Low Voltage

This ECG tracing, obtained from a patient with CAD, demonstrates sinus rhythm (rate: 60 beats/min) and incomplete right bundle branch block (RBBB) associated with posterior myocardial ischemia. Posterior myocardial ischemia is diagnosed on the basis of tall and upright T waves in leads V_1 through V_3 in the presence of RBBB. In other words, the primary T-wave change (the ischemic T-wave change) replaced the secondary T-wave change (biphasic to inverted T wave) because of RBBB in leads V_1 through V_3 (see Case 19). The term *low voltage* is used when a sum of the QRS voltage (positive as well as negative component) in leads I, II, and III is I5 mm or less.

Case 58 Diaphragmatic—Posterolateral Myocardial Ischemia with Marked Sinus Bradycardia

This ECG tracing was taken on a patient with exertional chest pain. The cardiac rhythm is markedly slow sinus bradycardia with a rate of 38 beats/min.

Abnormal ECG finding is manifested by inverted T waves in leads II, III, aVF, and V_5 through V_6 associated with tall upright T waves in leads V_1 through V_3 indicating diaphragmatic–posterolateral myocardial ischemia (see Case 55). In addition, diaphragmatic MI is a possibility.

Case 59 Coronary Artery Spasm Manifested by Diaphragmatic—Posterolateral Subepicardial Injury

A 41-year-old man was admitted to the hospital because of recurrent chest pain even at rest. (A) ECG obtained on admission while he was complaining of chest pain; (B) ECG taken several hours later when pain had subsided. In A there is a marked elevation of the ST segment in leads II, III, aVF, V₅, and V₆, accompanied by ST segment depression in leads V₁ through V₃. These findings indicate diaphragmatic-posterolateral subepicardial injury. In B, however, the ECG is completely normal. This type of transient ST segment elevation has been termed "variant angina pectoris," "atypical angina," or "Prinzmetal's angina." The basic rhythm is sinus in both tracings. The fundamental mechanism responsible for this transient ST segment elevation is thought to be coronary artery spasm.

ECG FINDINGS IN SUBEPICARDIAL INJURY

- Horizontal to upsloping ST segment elevation (may involve many leads)
- Transient ST segment elevation often due to coronary artery spasm
- Subepicardial injury potentially may lead to acute MI

Case 60 Marked Anterior Subepicardial Injury due to Coronary Artery Spasm and a Ventricular Premature Contraction

This ECG tracing was recorded on a 74-year-old man who was admitted to the coronary care unit for the evaluation of recurrent chest pain. The cardiac rhythm is sinus bradycardia with a rate of 57 beats/min, and there is a VPC in the rhythm strip (lead II). It is apparent that the ST segment is markedly elevated in the entire precordial leads, which is diagnostic of diffuse anterior subepicardial injury (see Case 59). Later, the diagnosis of coronary artery spasm was confirmed by coronary arteriography.

Case 61 Diaphragmatic—Posterolateral Subepicardial Injury due to Coronary Artery Spasm

This ECG tracing was recorded on a 57-year-old man who had been suffering from recurrent chest

pain. The cardiac rhythm is sinus, with a rate of 62 beats/min. The diagnosis of diaphragmatic-posterolateral subepicardial injury can be made on the basis of marked ST segment elevation in leads II, III, aVF, and V_5 through V_6 associated with ST segment depression in leads V_1 through V_2 (see Case 59). Again, coronary artery spasm was confirmed later by coronary arteriography.

Case 62 Diffuse Anterior Subepicardial Injury due to Coronary Artery Spasm and Bifascicular Block Consisting of Right Bundle Branch Block and Left Anterior Hemiblock

This ECG tracing was taken on a 79-year-old man who complained of severe substernal chest pain. The cardiac rhythm is sinus with a rate of 68 beats/min. There are several ECG abnormalities that include bifascicular block (BFB) consisting of RBBB and left anterior hemiblock (LAHB) associated with diffuse anterior subepicardial injury (see Cases 23 and 59). Note that the ST segment elevation is pronounced in all precordial leads. Coronary artery spasm should be strongly considered under these circumstances.

Case 63 Diffuse Anterior Subendocardial Injury with Probable Diaphragmatic Myocardial Infarction and Low Voltage

This ECG tracing was taken on a patient with severe chest pain of acute onset. The cardiac rhythm is sinus, with a rate of 78 beats/min. The obvious marked ST segment depression in leads V_1 through V_6 is indicative of severe anterior subendocardial injury. In addition, diaphragmatic MI must be considered; low voltage of the QRS complexes is also noted (see Case 57). Non-Q wave anterior MI should be strongly suspected in view of marked ST segment depression in all precordial leads.

ECG FINDINGS IN SUBENDOCARDIAL INJURY

- Horizontal to downsloping ST segment depression (may involve many leads)
- Persisting ST segment depression possibly indicative of non-Q wave MI (subendocardial MI)
- No pathologic Q wave

Case 64 Inverted U Waves Indicative of Diaphragmatic—Lateral Myocardial Ischemia and Left Ventricular Hypertrophy by Voltage

This ECG tracing was taken on a patient with hypertension and CAD. The cardiac rhythm is sinus bradycardia with a rate of 50 beats/min. The

inverted U wave (indicated by arrows) in leads V_4 through V_6 are indicative of lateral myocardial ischemia. Other ECG abnormalities include diaphragmatic myocardial ischemia and left ventricular hypertrophy (LVH) by voltage (see Case II).

Case 65 Extensive Anterior Myocardial Infarction with Prolonged QT Interval

This ECG tracing was obtained from a patient who was admitted to the coronary care unit because of acute chest pain. The cardiac rhythm is sinus bradycardia, with a rate of 56 beats/min. The very large inverted T waves with prolonged QT interval involve practically every ECG lead in the presence of recent extensive anterior MI. It has been shown that very large inverted T waves are relatively common ECG findings in patients with recent MI (from I week to I0 days after the MI).

DIAGNOSTIC CRITERIA OF ABNORMAL Q WAVES

- Q wave width of 0.04 second or more
- Q wave depth of 25% of the R wave amplitude or more

DIAGNOSTIC CRITERIA OF MI (FROM THE VIEWPOINT OF INVOLVED LOCATION)

- Anteroseptal MI: Q or QS waves in leads V₁ through V₃
- Localized anterior MI: Q or QS waves in leads
 V₂ through V₄
- Anterolateral MI: Q or QS waves in leads V₄ through V₆
- High lateral MI: Q or QS waves in leads I and aVL
- Extensive (massive) anterior MI: Q or QS waves in leads I, aVL, and V₁ through V₆
- Diaphragmatic (inferior) MI: Q waves in leads II, III, and aVF (QS waves are not reliable criteria)
- Posterior MI: Tall (or relatively tall) R waves in leads V₁ through V₃

Case 66 Acute Anteroseptal Myocardial Infarction

This ECG tracing was taken in the emergency room on a patient with acute chest pain of recent onset. It shows sinus rhythm (rate: 80 beats/min) and very acute anteroseptal MI with marked anteroseptal subepicardial injury (see Case 65).

Case 67 Acute Diaphragmatic Myocardial Infarction with Posterolateral Subepicardial Injury

This ECG tracing was recorded from a patient who was brought to the emergency room because of

severe chest pain. The cardiac rhythm is sinus, with a rate of 94 beats/min. The diagnosis of acute diaphragmatic (inferior) MI can be readily made using the conventional criteria (see Case 65). In addition, posterolateral subepicardial injury can be diagnosed on the basis of the ST segment depression in leads V_1 through V_2 with ST segment elevation in leads V_4 through V_6 (see Case 59).

Case 68 Recent Diaphragmatic—Posterior Myocardial Infarction with Lateral Myocardial Ischemia

This ECG tracing was taken on a patient with severe chest discomfort. The cardiac rhythm is sinus with a rate of 70 beats/min. The diagnosis of recent diaphragmatic–posterior MI can be entertained using the conventional criteria (see Case 65). In addition, lateral myocardial ischemia can be diagnosed on the basis of inverted T waves in leads V_5 and V_6 .

Case 69 High Lateral Myocardial Infarction with Possible Posterior Myocardial Infarction and Lateral Myocardial Ischemia

This ECG tracing was recorded from a patient who had suffered a heart attack 2 weeks prior to the recording. The cardiac rhythm is sinus, with a rate of 70 beats/min. The ECG abnormalities include high lateral MI, possible posterior MI, and lateral myocardial ischemia using the conventional diagnostic criteria (see Case 65).

Case 70 Posterolateral Myocardial Infarction Including High Lateral Myocardial Infarction

This ECG tracing was recorded from a patient who had suffered from a heart attack 3 weeks prior to the recording. The cardiac rhythm is sinus bradycardia, with a rate of 58 beats/min. Using the conventional criteria, the diagnosis of subacute posterolateral MI including the high lateral wall can be made without any difficulty (see Case 65).

Case 71 Diaphragmatic Posterolateral Myocardial Infarction Associated with Marked Sinus Tachycardia

The cardiac rhythm is marked sinus tachycardia, with a rate of I47 beats/min. The diagnosis of acute diaphragmatic posterolateral MI (DPLMI) can be made without any difficulty using the conventional criteria (see Case 65). Note that the pathologic Q waves in leads II, III, aVF, and V₅ through V₆ with

ST segment elevation and relatively tall R waves in leads V_1 through V_2 with ST segment depression are diagnostic indicators of DPLMI.

Case 72 Acute Extensive Anterior Myocardial Infarction Associated with Diaphragmatic Myocardial Infarction and Low Voltage

The cardiac rhythm is marked sinus tachycardia, with a rate of 142 beats/min. The diagnosis of acute extensive anterior MI can be made readily using the conventional criteria (see Case 65). In addition, recent diaphragmatic MI is also evident (see Case 65). Note the marked ST segment elevation in practically all precordial leads with diagnostic (pathologic) Q waves. The ST segment elevation in leads II, III, and aVF is less pronounced. The QRS amplitude in limb leads is small, meaning low voltage (see Case 57). Low voltage is very common in patients with acute MI, especially when the MI is massive.

Case 73 Extensive Anterior Myocardial Infarction with Right Bundle Branch Block and Left Atrial Enlargement

This ECG tracing was taken on a patient with a known CAD. The cardiac rhythm is sinus bradycardia, with a rate of 54 beats/min. The ECG abnormalities are RBBB, extensive anterior MI including high lateral wall and left atrial enlargement (LAE) (see Cases 6, 19, and 65).

Case 74 Diaphragmatic—Posterior Myocardial Infarction with Right Bundle Branch Block, Anterior Myocardial Ischemia, and Anterior Subendocardial Injury

This patient was admitted to the coronary care unit because of severe chest discomfort. The cardiac rhythm is sinus, with a rate of 100 beats/min. The diagnosis of recent diaphragmatic MI can be readily made on the basis of the large pathologic Q waves in leads II, III, and aVF. On the other hand, posterior MI may not be recognized by inexperienced readers because of coexisting RBBB. It should be noted that the initial R wave in lead V1 is much taller than for usual RBBB. This tall initial R wave in lead V₁ represents posterior MI (see Case 65). In addition, diffuse anterior myocardial ischemia as well as anterior subendocardial injury are diagnosed on the basis of inverted T waves with downsloping ST segment depression involving practically all precordial leads (see Case 63).

Case 75 Diaphragmatic—Posterolateral Myocardial Infarction with Right Bundle Branch Block

This ECG tracing was taken on a patient who was seen at the cardiac clinic for follow-up care. The cardiac rhythm is sinus, with a rate of 98 beats/min. The ECG abnormalities include RBBB and diaphragmatic-posterolateral MI, according to the conventional criteria (see Cases 19 and 65).

Case 76 Recent Diaphragmatic Myocardial Infarction with Left Bundle Branch Block

A 46-year-old obese and hypertensive woman was admitted to the coronary care unit because of acute chest pain. The cardiac rhythm is sinus, with a rate of 82 beats/min. The diagnosis of left bundle branch block (LBBB) is obvious, according to the conventional criteria (see Case 26). Another striking ECG abnormality, the deep and large Q waves in leads II, III, and aVF associated with significant ST segment elevation, is a diagnostic finding of recent diaphragmatic MI (see Case 65).

Case 77 Diffuse Myocardial Ischemia with Left Bundle Branch Block

A 71-year-old man with a long-standing hypertension was brought to the emergency room because of acute chest discomfort. The cardiac rhythm is sinus, with a rate of 74 beats/min. The diagnosis of LBBB can be made without any difficulty using the conventional criteria (see Case 26). Another ECG abnormality is symmetrically and deeply inverted T waves involving practically all leads, diffusely. This ECG finding represents diffuse myocardial ischemia (see Cases 55 and 65). Acute MI is strongly considered in these circumstances if this ECG abnormality persists more than 24 hours.

Case 78 Extensive Anterior Myocardial Infarction with Bifascicular Block Consisting of Right Bundle Branch Block and Left Anterior Hemiblock

This patient was admitted to the coronary care unit because of chest pain that lasted a few hours. The cardiac rhythm is sinus rhythm (rate: I00 beats/min) and with first-degree A-V block (PR interval: 0.22 second). The ECG abnormalities include BFB consisting of RBBB and LAHB associated with recent extensive anterior MI (see Cases 23 and 65).

Case 79 Recent Posterolateral Myocardial Infarction with Bifascicular Block Consisting of Right Bundle Branch Block and Left Anterior Hemiblock

A 73-year-old man was evaluated in the emergency room because of his chest pain of recent onset. The cardiac rhythm is sinus, with a rate of 100 beats/min. This ECG exhibits BFB consisting of RBBB and LAHB associated with recent posterolateral MI including high lateral wall involvement (see Cases 23 and 65). The diagnosis of high lateral MI can be entertained on the basis of the abnormal Q waves in leads I and aVL.

Case 80 Atrial Flutter-Fibrillation with Acute Extensive Anterior Myocardial Infarction and Low Voltage

This ECG tracing was recorded from a patient with severe chest pain of a few hours in duration associated with rapid heart action. The cardiac rhythm is atrial flutter-fibrillation with rapid ventricular response (ventricular rate: 115–134 beats/min). There are two major ECG abnormalities: RBBB and acute extensive anterior MI (see Cases 19 and 65). In addition, the QRS complex amplitude is markedly reduced in the limb leads, which is called low voltage (LV) (see Case 57).

Case 81 Acute Right Ventricular Myocardial

This ECG tracing was taken on a patient who was admitted to the coronary care unit because of acute chest pain. The cardiac rhythm is sinus, with a rate of 73 beats/min. It is important to recognize that the marked ST segment elevation in leads V_1 through V_3 is diagnostic of acute right ventricular MI. In right ventricular MI, the ST segment elevation is most pronounced in lead V_1 , and the ST segment elevation is progressively less evident toward leads V_2 and V_3 . Remember that abnormal Q waves are not present in right ventricular MI.

DIAGNOSTIC CRITERIA OF RIGHT VENTRICULAR MI

- ST segment elevation (I mm or more) in leads
 V₁ through V₃
- ST segment elevation (I mm or more) in leads V_1 and V_{3R} through V_{6R}
- ST segment elevation in leads V₁ through V₆ (marked in lead V₁ and least in lead V₆)
- Often associated with acute diaphragmatic MI of the left ventricle
- Reduction of R' wave amplitude in leads V₁ through V₂ with reduction of depth of S waves in leads V₄ through V₆ in the presence of RBBB

Case 82 Non-Q Wave Myocardial Infarction

This ECG tracing was taken on a patient with CAD. The cardiac rhythm is sinus, with a rate of 72 beats/min. It is obvious to recognize significant downsloping ST segment depression with biphasic to inverted T waves in practically every lead. The diagnosis of non-Q wave MI (subendocardial MI) is strongly considered when the above ECG finding persists more than 24 hours, provided that acute MI is clinically evident.

DIAGNOSTIC CRITERIA OF NON-Q WAVE MI

- Persisting ST segment depression (horizontal to downsloping) of 1 mm or more
- Persisting symmetric T-wave inversion (less reliable)
- Elevation of creatine phosphokinase (CPK) with MB isoenzyme
- No abnormal (pathologic) Q wave

Case 83 Non-Q Wave Myocardial Infarction with Sinus Tachycardia

This patient was brought to the emergency room because of severe chest pain of acute onset. The cardiac rhythm is marked sinus tachycardia with a rate of 132 beats/min. The diagnosis of acute non-Q wave MI (subendocardial MI) is entertained on the basis of marked downsloping ST segment depression with biphasic to inverted T waves involving practically every lead (see Case 82).

Case 84 Pseudo Anteroseptal Myocardial Infarction due to Wolff-Parkinson-White Syndrome, Type B

This ECG tracing was recorded from an 18-year-old girl with palpitations. The cardiac rhythm is sinus arrhythmia (rate: 76–125 beats/min). The diagnosis of Wolff-Parkinson-White (WPW) syndrome, type B can be made using the conventional criteria (a short PR interval with a delta wave involving every lead). Pseudo anteroseptal MI is produced because the delta waves are directed posteriorly and to the left in WPW syndrome, type-B (see Section 10).

PSEUDO MI PATTERNS

- WPW syndrome
- Idiopathic hypertrophic subaortic stenosis (IHSS)
- LBBB
- LVH
- LAHB
- Chronic obstructive pulmonary disease (COPD)
- Cardiomyopathy
- · Chest deformity

 Miscellaneous conditions (normal variants, pericarditis, pulmonary embolism, mitral valve prolapse syndrome, central nervous system disorders, hyperkalemia)

Case 85 Pseudo Posterior and High Lateral Myocardial Infarction due to Wolff-Parkinson-White Syndrome, Type A

This ECG tracing was recorded from a healthy young woman with WPW syndrome, type A. The cardiac rhythm is sinus bradycardia (rate: 56 beats/min). Pseudo posterior and high lateral MI is produced in this recording, as the delta waves are directed anteriorly and to the right in WPW syndrome, type A (see Section 10).

Case 86 Pseudo Anteroseptal Myocardial Infarction due to Left Bundle Branch Block

The cardiac rhythm is a normal sinus rhythm with a rate of 93 beats/min. Pseudo anteroseptal MI is produced by LBBB. In LBBB, leads V_1 through V_3 may show very small r waves or even no R waves (QS complexes), depending upon the direction of the ventricular septal activation (see Case 26). Inexperienced readers often may misdiagnose anteroseptal MI under this circumstance.

Case 87 Pseudo Posterior Myocardial Infarction due to Idiopathic Hypertrophic Subaortic Stenosis

This ECG tracing, obtained from a young patient with IHSS, demonstrates pseudo posterior M1 because of 1HSS (Cases 65 and 84). The cardiac rhythm is sinus, with a rate of 60 beats/min.

Case 88 Pseudo Anteroseptal Myocardial Infarction due to Left Ventricular Hypertrophy

The underlying cardiac rhythm is atrial fibrillation (AF) with advanced A-V block (ventricular rate: 60–75 beats/min). Pseudo anteroseptal MI is produced by marked LVH (see Case 11). Pseudo anteroseptal or anterior MI is often produced whenever there is markedly increased electrical potential posteriorly and to the left as a result of severe LVH, as small r waves or even no R waves (QS complexes) are observed in leads V1 through V3 (see Case 11).

Case 89 Pseudo Anteroseptal Myocardial Infarction due to Central Nervous System Disorders Associated with High Left Ventricular Voltage

A young woman was brought to the emergency room in a comatose state. The cardiac rhythm is sinus arrhythmia (rate: 75–105 beats/min). Among the striking ECG abnormalities is a marked ST segment elevation with inverted and broad T waves involving many leads, associated with QS waves in leads V₁ through V₃. In this case, pseudo anteroseptal M1 with diffuse subepicardial injury pattern is produced by her subarachnoid hemorrhage (see Cases 59 and 65). In addition, high left ventricular voltage (HLVV) is diagnosed (see Cases 3 and 11).

Section 5

Sinus Arrhythmias

Case 90 Sinus Tachycardia

This ECG tracing was recorded on a healthy young person. The cardiac rhythm is sinus tachycardia with a rate of I40 beats/min, and the ECG is within normal limits. The diagnostic criteria of normal sinus rhythm (NSR) were described earlier (see Case 1). The sinus rhythm faster than 100 beats/min is called *sinus tachycardia*.

Case 91 Sinus Bradycardia

These ECG rhythm strips are obtained from a healthy individual. The cardiac rhythm is sinus bradycardia with a rate of 55 beats/min, and the ECG is within normal limits. The sinus rhythm slower than 60 beats/min is terminal sinus bradycardia.

Case 92 Sinus Arrhythmia

ECG tracing from a healthy young girl. The cardiac rhythm is sinus bradycardia and sinus arrhythmia (rate: 50–75 beats/min); otherwise, it is within normal limits. The numbers represent hundredths of a second. The term, *sinus arrhythmia* is used when the shortest and the longest sinus cycles vary more than 0.12 second. Sinus arrhythmia often coexists with sinus bradycardia, especially among young healthy people.

Case 93 Sinus Arrhythmia with Wandering Atrial Pacemaker

ECG tracing from a young healthy child. The cardiac rhythm is marked sinus arrhythmia with wandering atrial pacemaker to the atrioventricular (A-V) node (rate: 53–65 beats/min). Note that the P wave configuration varies from upright to inverted P waves. Wandering atrial pacemaker often coexists with sinus arrhythmia, particularly among healthy young adults and children.

Case 94 Sinus Bradycardia with Intermittent Atrioventricular Junctional Escape Rhythm Producing Incomplete Atrioventricular Dissociation

Leads II-a to II-d are continuous; the arrows indicate sinus P waves. The underlying cardiac rhythm is markedly slow sinus bradycardia (atrial rate: 44 beats/min), but the dominant rhythm is A-V junctional escape rhythm (A-V JER) (ventricular rate: 50 beats/min) producing incomplete A-V dissociation. Note the frequent ventricular captured beats (normally conducted sinus beats, marked CB).

Case 95 Wenckebach Atrioventricular Block with Intermittent Sinus Arrest Leading to Intermittent Atrioventricular Junctional Escape Rhythm

Arrows indicate sinus P waves. The underlying cardiac rhythm is sinus (rate: 75 beats/min), with slowly progressing Wenckebach A-V block. A-V JER (marked N) occurs intermittently as a result of a long sinus arrest (4.80 seconds). This ECG finding is a manifestation of sick sinus syndrome (SSS). Sinus arrest means that the sinus node fails to produce any cardiac impulse, and the underlying disease process is usually dysfunctioning sinus node, meaning SSS (see Case 96).

Case 96 Sinus Arrest with Intermittent Atrioventricular Junctional Escape Rhythm Indicative of Sick Sinus Syndrome

Leads II-a and II-b are continuous, and arrows indicate sinus P waves. The underlying cardiac rhythm is sinus bradycardia (rate: 58 beats/min), but a very slow A-V JER (marked N) occurs as a result of sinus arrest (5.25 seconds). This ECG finding is a manifestation of an advanced SSS.

ECG MANIFESTATIONS OF SSS

- Marked and persisting sinus bradycardia (not drug-induced)
- Sinus arrest or sinoatrial (S-A) block (see Cases 95 to 99)
- Drug-resistant sinus bradyarrhythmias
- Long pause after an atrial premature contraction (APC)
- Prolonged sinus node recovery time by atrial pacing
- Chronic atrial fibrillation (AF; or less commonly atrial flutter) with advanced A-V block
- A-V JER with or without slow and unstable sinus activity

- Carotid sinus syncope (see Case 97)
- Failure of restoration of sinus rhythm following cardioversion
- Brady-tachyarrhythmia syndrome (BTS) (see Cases I03 and I04)
- Any combination of the above

Case 97 Sick Sinus Syndrome Manifested by Carotid Sinus Syncope, Sinus Arrest, Atrioventricular Junctional Escape Beats, and Ventricular Standstill

These cardiac rhythm strips were obtained from an elderly patient who had frequent episodes of syncope. Leads II-a to II-c are continuous, and arrows indicate sinus P waves. The underlying cardiac rhythm is sinus (rate: I00 beats/min), but a very unstable and slow A-V JER occurs intermittently as a result of a long sinus arrest, producing a long ventricular standstill. These ECG findings are the result of a severe reaction to carotid sinus stimulation, called *carotid sinus syncope*. A permanent artificial pacemaker was implanted on this patient. Various manifestations of SSS were described previously (see Case 96).

Case 98 Sick Sinus Syndrome Manifested by Sinus Arrest and Intermittent Ventricular Escape Rhythm, Producing Incomplete Atrioventricular Dissociation and Left Ventricular Hypertrophy

This ECG tracing was taken on a 70-year-old woman who had occasional episodes of near syncope. Arrows indicate sinus P waves. The underlying cardiac rhythm is sinus (atrial rate: 72 beats/min), but ventricular escape rhythm (VER; see the first, second, third, sixth, and seventh beats in rhythm strip lead II, rate 45 beats/min) occurs intermittently, leading to incomplete A-V dissociation. Note the occasional ventricular fusion beats (e.g., the first and third beats in the rhythm strip lead II). The fundamental mechanism for the production of VER is sinus arrest (see Case 96). SSS is the underlying disorder. The diagnosis of LVH is made using the conventional criteria (see Case II).

Case 99 Sick Sinus Syndrome Manifested by 2:1 Sinoatrial Block

The underlying cardiac rhythm is sinus (rate: 74 beats/min), but type II 2: I S-A block occurs intermittently. Note that the long P-P interval is twice the basic P-P cycle, indicating that every other sinus impulse fails to conduct to the atria as a result of a block in the S-A junction. (The numbers represent

hundredths of a second.) S-A block is one of the less common manifestations of SSS (see Case 96).

DIAGNOSTIC CRITERIA OF TYPE II S-A BLOCK

- There is an occasional absence of one or more P waves of the sinus origin.
- The P-P interval due to S-A block is exactly (or almost exactly) a multiple of the basic P-P cycle.
- Type II S-A block may occur periodically at regular or irregular intervals.
- One or more A-V junctional or ventricular escape beats may occur in S-A block.

Case 100 Wenckebach Sinoatrial Block Associated with Wenckebach Atrioventricular Block due to Acute Diaphragmatic Myocardial Infarction

These cardiac rhythm strips were obtained from a patient with acute heart attack. Leads II-a to II-c are continuous. The cardiac rhythm shows sinus rhythm (indicated by the arrows) with 3:2 Wenckebach A-V block as well as Wenckebach S-A block. The characteristic features of Wenckebach A-V block are disturbed by a coexisting Wenckebach S-A block; the periodic occurrence of the expected blocked P waves is not shown except for only one blocked P wave in lead II-a (marked P) because of the coexisting S-A block. It is not uncommon to observe the coexisting S-A and A-V block in the same patient who has acute diaphragmatic MI, as shown in this case. Diagrammatic illustration to explain A-V block and S-A block is shown in this ECG (S, sinus node; S-A, sinoatrial; A, atria; A-V, atrioventricular; V, ventricles).

DIAGNOSTIC CRITERIA OF WENCKEBACH (TYPE I) S-A BLOCK

- Progressive shortening of the sinus P-P intervals is followed by a long pause (long P-P interval).
- Long P-P interval is less than two basic P-P cycles.
- Wenckebach S-A block may occur periodically at regular or irregular intervals.
- One or more A-V junctional or ventricular escape beats may occur.

Case 101 Wenckebach Sinoatrial Block Associated with Wenckebach Atrioventricular Block

This ECG tracing was taken on a 72-year-old woman. The cardiac rhythm diagnosis is sinus rhythm with predominantly 3:2 Wenckebach S-A block associated with a slowly progressing Wenckebach A-V block (see Case 100 and Section 9). Diagrammatic illustration to explain the mechanism of

S-A block is found in Case 100. The diagnostic criteria of Wenckebach S-A block are described previously (see Case 100).

Case 102 Sick Sinus Syndrome Manifested by Sinus Bradycardia and Intermittent Atrial Flutter with Advanced Atrioventricular Block

This ECG tracing was taken on a patient with near syncope. The underlying cardiac rhythm is markedly unstable and slow sinus bradycardia, but atrial flutter with advanced A-V block occurs intermittently. This ECG finding is a manifestation of SSS (see Case 96).

Case 103 Advanced Sick Sinus Syndrome Manifested by Brady-Tachyarrhythmia Syndrome Consisting of Marked and Unstable Sinus Bradycardia, Sinus Arrest, Intermittent Atrial FlutterFibrillation, and Ventricular Premature Contractions

The Holter monitor ECG rhythm strips A to E are not continuous. The underlying cardiac rhythm is markedly unstable sinus mechanism (indicated by arrows) with sinus arrest and intermittent atrial flutter-fibrillation. These ECG findings represent brady-tachyarrhythmia syndrome (BTS), which is a form of advanced SSS (see Case 96). Note the frequent bizarre beats that represent aberrant ventricular conduction (AVC) as a result of Ashman's phenomenon (see Section 6). There are also occasional ventricular premature contractions (VPCs; see the last beat in strip A).

Case 104 Sick Sinus Syndrome Manifested by Brady-Tachyarrhythmia Syndrome Consisting of Marked Sinus Bradycardia, Sinus Arrest and Intermittent Atrial Fibrillation, and Atrioventricular Junctional Escape Beats with Ventricular Escape Beat as well as Ventricular Premature Contraction

The Holter monitor ECG rhythm strips A to D are not continuous. The underlying cardiac rhythm is markedly unstable sinus mechanism (indicated by arrows) with intermittent sinus arrest and intermittent A-V junctional escape beats (JEBs; marked N), VPCs (marked V), as well as the ventricular escape beat (VEB; marked X). These ECG findings represent BTS, which is a manifestation of advanced SSS (see Case 96).

Case 105 Ventriculophasic Sinus Arrhythmia Associated with Complete Atrioventricular Block and Atrioventricular Junctional Escape Rhythm

The underlying cardiac rhythm is sinus (atrial rate: 90 beats/min), but none of the sinus impulses are conducted to the ventricles as a result of complete A-V block. Thus, the cardiac rhythm diagnosis is sinus rhythm with A-V JER (ventricular rate: 50 beats/min) due to complete A-V block (see Section 9). In addition, ventriculophasic sinus arrhythmia (VPSA) is diagnosed because the P-P interval including a QRS complex is shorter that the P-P interval without a QRS complex. It has been shown that VPSA is observed in 30% of complete A-V block cases. (The numbers illustrated represent hundredths of a second.)

Section 6

Atrial Arrhythmias

Case 106 Atrial Premature Contractions Causing Atrial Bigeminy Associated with Acute Anteroseptal Myocardial Infarction and Right Bundle Branch Block

These ECG rhythm strips were obtained from a patient with acute chest pain of a few hours' duration. The underlying cardiac rhythm is sinus, but there are frequent atrial premature contractions (APCs) leading to atrial bigeminy (indicated by arrows). Other ECG abnormalities include acute anteroseptal myocardial infarction (MI) and right bundle branch block (RBBB) (see Cases 19 and 65).

DIAGNOSTIC CRITERIA OF APC

- A premature ectopic P wave arises from any portion in the atria.
- The premature P wave is usually upright in lead II and inverted in lead aVR.
- The premature P wave is followed by a normal (narrow) QRS complex unless there is aberrant ventricular conduction (AVC), left bundle branch block (LBBB), or RBBB.
- The premature P wave may not be followed by a QRS complex (blocked APC).
- There is a constant coupling interval.
- APC often leads to atrial tachycardia, flutter, or fibrillation.

Case 107 Atrial Premature Contractions Causing Atrial Bigeminy Associated with Aberrant Ventricular Conduction

The underlying cardiac rhythm is sinus, but there are frequent APCs (indicated by arrows) producing atrial bigeminy. All of the APCs demonstrate AVC (RBBB pattern) as a result of a short coupling interval as well as Ashman's phenomenon. APCs with AVC superficially mimic ventricular premature contractions (VPCs).

THE THREE CAUSES OF ABERRANT VENTRICULAR CONDUCTION (AVC)

- Ashman's phenomenon
- Short coupling interval
- Very rapid ventricular rate (faster than I60–I80 beats/min)

ASHMAN'S PHENOMENON

- The longer the ventricular cycle (R-R interval), the longer is the refractory period following it; the shorter the ventricular cycle, the shorter is the refractory period.
- Ashman's phenomenon may be recognized in any cardiac rhythm when AVC occurs following a long ventricular cycle.
- Ashman's phenomenon is the most common cause of AVC.

Case 108 Atrial Premature Contractions with Aberrant Ventricular Conduction (Multiformed)

The underlying cardiac rhythm is sinus (rate: 60 beats/min), but there are frequent APCs (indicated by narrows). It is interesting to note that some APCs show LBBB pattern (marked L) whereas others exhibit RBBB pattern (marked R). AVC and Ashman's phenomenon are described previously (see Case 107).

Case 109 Atrial Premature Contractions with Multiformed Aberrant Ventricular Conduction Leading to Paroxysmal Atrial Tachycardia Associated with Aberrant Atrial Conduction (Chung's Phenomenon)

The underlying cardiac rhythm is sinus (rate: 64 beats/min), but there are frequent APCs (indicated by arrows). Some APCs with AVC show LBBB pattern (marked L), whereas others exhibit RBBB pattern (marked R). In addition, there are atrial group beats (in lead II-a) and paroxysmal atrial tachycardia with a rate of I52 beats/min (in lead II-b). Another interesting ECG finding is the

deformed sinus P wave (marked X) following an APC in lead V_2 as a result of Chung's phenomenon; leads II-a and II-b are not continuous. Chung's phenomenon (aberrant atrial conduction) is defined as an alteration of the sinus P wave configuration following any ectopic beat (commonly an APC).

Case 110 Blocked (Non-Conducted) Atrial Premature Contractions

The underlying cardiac rhythm is sinus (rate: 65 beats/min), but there are frequent APCs (indicated by arrows). It is interesting to note that some APCs are not followed by QRS complexes (blocked APCs) leading to ventricular pause. Blocked APCs superficially mimic various other arrhythmias including marked sinus arrhythmia, sinus bradycardia, sinus arrest, sinoatrial (S-A) block, and second-degree atrioventricular (A-V) block (see Sections 5 and 9).

THE FOUR MAJOR CAUSES OF VENTRICULAR PAUSE

- Blocked (non-conducted) APC (most common cause)
- Second-degree or higher A-V block
- Sinus arrest
- S-A block

Rarely, a blocked reciprocal beat (echo beat) or a blocked A-V junctional premature contraction (JPC) may cause a ventricular pause.

Case 111 Atrial Premature Contractions, Some Blocked and Some Aberrantly Conducted with Atrial Bigeminy, Ventricular Premature Contractions, and Possible Anteroseptal Myocardial Infarction

This ECG tracing was taken on a 63-year-old man with coronary artery disease (CAD). The underlying cardiac rhythm is sinus (rate: 92 beats/min), but there are frequent APCs (indicated by arrows); some are blocked, whereas others are aberrantly conducted to the ventricles (see Cases 107 and 110). In addition, there are frequent VPCs (see the first and seventh beats in the rhythm strip lead II). The diagnosis of old anteroseptal MI is a possibility (see Case 65).

Case 112 Atrial Premature Contractions, Some Interpolated, Some Blocked, and Some Aberrantly Conducted Causing Atrial Bigeminy

This ECG tracing, which was obtained from a 20-year-old, apparently healthy girl, was discussed during a weekly ECG conference because of its interesting ECG findings. The underlying cardiac

rhythm is sinus, but there are frequent APCs (indicated by arrows) producing atrial bigeminy. Some APCs are interpolated (the first two APCs), and some APCs are aberrantly conducted to the ventricles (the first two APCs also); the remaining APCs are not conducted to the ventricles (blocked or nonconducted APCs) in the rhythm strip lead II (see Cases 107 and 110). When dealing with an interpolated APC, a protection block of the sinus node has to be considered. Many inexperienced readers may not recognize blocked APCs, resulting in an erroneous diagnosis. In addition, APCs with AVC may be easily mistaken for VPCs. Careful analysis of each ECG tracing—particularly the recognition of all ectopic P waves—is essential to make the correct diagnosis.

Case 113 Atrial Premature Contractions with Blocked Atrial Bigeminy

This ECG tracing was taken on a 43-year-old man who was referred to the cardiac clinic because of his somewhat unusual cardiac arrhythmia. The underlying cardiac rhythm is sinus, but ectopic premature P waves (indicated by arrows) occur on every other beat. There are frequently blocked APCs (indicated by arrows) producing a blocked atrial bigeminy. All APCs are not conducted to the ventricles, because the ectopic atrial impulses are conducted to the A-V junction during its absolute refractory period. Many inexperienced readers may not be able to recognize blocked atrial bigeminy, and an erroneous diagnosis (e.g., sinus bradycardia) may be entertained. In this case, the deformed T waves are a clue in recognizing blocked APCs (see Case 110).

Case 114 Sick Sinus Syndrome Manifested by Marked Sinus Bradycardia, Sinus Arrest, Atrioventricular Junctional Escape Beats, Atrial Premature Contraction, and Intermittent Atrial Flutter Associated with Left Ventricular Hypertrophy

A 67-year-old woman was examined at the clinic for the evaluation of a near syncope associated with irregular and slow pulse. The underlying cardiac rhythm is sinus arrhythmia (rate: 70 beats/min), and there is an APC (indicated by arrow). In addition, her cardiac rhythm changes from atrial flutter with 2:1 A-V conduction (the first half of the 12-lead ECG) to sinus arrest and occasional A-V JEBs (the last half of the 12-lead ECG). These cardiac rhythm disorders are manifestations of SSS (see Case 96). Using the conventional criteria, a diagnosis of

left ventricular hypertrophy (LVH) can be considered (see Case 11).

Case 115 Atrial Premature Contractions with Chung's Phenomenon (Aberrant Atrial Conduction)

Arrows indicate ectopic P waves. The underlying cardiac rhythm is sinus (rate: 64 beats/min), and there are occasional APCs (indicated by arrows). It is interesting to note that the sinus P wave (marked X) immediately following an APC is deformed. This unusual ECG finding is termed Chung's phenomenon (aberrant atrial conduction) (see Case 109).

Case 116 Paroxysmal Atrial Tachycardia with Aberrant Ventricular Conduction

The Holter monitor ECG rhythm strips A to C are not continuous. The underlying cardiac rhythm is sinus, but paroxysmal atrial tachycardia (PAT) occurs intermittently with a rate of 215 beats/min. Multiformed aberrant ventricular conduction (AVC) during PAT occurs with the same ventricular rate (see Case 107).

Case 117 Atrial Tachycardia with 2:1 Atrioventricular Block and Left Ventricular Hypertrophy

Arrows indicate P waves. The cardiac rhythm is atrial tachycardia (atrial rate: 214 beats/min) with 2:1 A-V block (ventricular rate: 107 beats/min). Note that every other P wave is conducted to the ventricles. The diagnosis of LVH is established using the conventional criteria (see Case 11).

Case 118 Paroxysmal Atrial Tachycardia with Wenckebach Atrioventricular Block and Ventricular Premature Contractions due to Digitalis Intoxication

Leads 11-a and I1-b are continuous, and arrows indicate P waves. The cardiac rhythm is PAT (atrial rate: 188 beats/min) with Wenckebach A-V block and intermittent 2:1 A-V block. In addition, there are multifocal VPCs (marked V). Digitalis intoxication is responsible for the production of these arrhythmias.

Case 119 Multifocal Atrial Tachycardia with a Ventricular Premature Contraction Associated with Left Anterior Hemiblock and Right Atrial Enlargement (P-Pulmonale)

This ECG tracing was obtained from an 81-year-old man with chronic obstructive pulmonary disease

(COPD). The cardiac rhythm is multifocal atrial tachycardia (MAT; rate: 155 beats/min) and a VPC (the ninth beat). Other ECG abnormalities include LAHB and RAE (P-pulmonale) (see Cases 8 and 36). Note that MAT is manifested by varying P wave configurations with varying P-P cycles and varying P-R intervals.

DIAGNOSTIC CRITERIA OF MAT

- Two or more ectopic P waves with different configurations and two or more different P-P cycles
- Atrial rate of 100–250 beats/min (occasionally less than 100 beats/min)
- Isoelectrical line between P-P intervals
- Varying P-R intervals and A-V block of varying degree (non-conducted ectopic P waves)

Case 120 Multifocal Atrial Tachycardia with Aberrant Ventricular Conduction

Arrows indicate P waves. The cardiac rhythm is MAT (rate: 180–230 beats/min) with frequent AVC that occurs consecutively (see Cases 107 and 119). Consecutively occurring AVC closely simulates VPCs and a short run of ventricular tachycardia (VT). AVC occurs because of the very rapid rate, and it is initiated by Ashman's phenomenon (see Case 107). Note that each bizarre QRS complex is preceded by an ectopic P wave. Thus, the possibility of VPCs or VT is excluded.

Case 121 Atrial Fibrillation with Acute Extensive Anterior Myocardial Infarction, Probable Diaphragmatic Myocardial Infarction, and Low Voltage

A 71-year-old man was brought to the emergency room because of severe chest pressure associated with a very rapid heart action. The cardiac rhythm is atrial fibrillation (AF) with a very rapid ventricular response (ventricular rate: 153–173 beats/min). Acute extensive anterior M1 can be diagnosed using the conventional criteria (see Case 65). In addition, a possibility of diaphragmatic M1 can be considered. Note that the QRS amplitude is markedly reduced; this is referred to as *low voltage* (LV) (see Case 57).

Case 122 Atrial Flutter-Fibrillation with Aberrant Ventricular Conduction

The cardiac rhythm is atrial flutter-fibrillation (a mixed form of atrial fibrillation and flutter) with two aberrantly conducted beats due to Ashman's phenomenon (see Case 107). VPCs are closely simulated, but the lack of any ventricular pause following the bizarre beat is the evidence against a VPC.

Case 123 Atrial Fibrillation with Consecutive Aberrant Ventricular Conduction

The cardiac rhythm is atrial fibrillation with very rapid ventricular response (rate: 180–210 beats/min) and consecutively occurring aberrant ventricular conduction involving 10 beats initiated by Ashman's phenomenon. A short run of nonsustained VT is closely simulated, but the possibility of VT is excluded on the basis of a lack of ventricular pause following the bizarre beats in addition to the presence of Ashman's phenomenon (see Case 107 and Section 8).

Case 124 Atrial Flutter with 2:1 Atrioventricular Conduction

The cardiac rhythm is atrial flutter (atrial rate: 334 beats/min) with 2:1 A-V conduction (ventricular rate: 167 beats/min). Notice the sawtooth appearance of the atrial flutter waves. Every other flutter wave is conducted to the ventricles. Atrial flutter with 2:1 A-V conduction is a physiologic phenomenon because the A-V node has a long refractory period. Thus, the term, "2:1 A-V response" is used in this circumstance (atrial flutter with "2:1 A-V block" is erroneous).

Case 125 Atrial Flutter with 1:1 Atrioventricular Conduction and Left Anterior Hemiblock

The cardiac rhythm is atrial flutter with 1:1 A-V conduction leading to extremely rapid ventricular rate of 300 beats/min. Occurrences of atrial flutter with 1:1 A-V conduction are extremely rare in our experience. However, 1:1 A-V conduction in atrial flutter may occur under certain conditions, including during postoperative periods, and with hyperthyroidism, severe emotional stress, vigorous physical activity, and WPW syndrome (see Section 10). In the majority of cases, atrial flutter exhibits 2:1 A-V conduction (see Case 124). The diagnosis of left anterior hemiblock (LAHB) is made using the conventional criteria (see Case 36).

Case 126 Atrial Flutter with 3:1 Atrioventricular Block with Incomplete Right Bundle Branch Block and Left Anterior Hemiblock

The cardiac rhythm is atrial flutter with 3:1 A-V block (ventricular rate: 74 beats/min). Other ECG abnormalities include incomplete RBBB and LAHB leading to a partial BFB (see Case 23). In addition,

RAE is considered in view of the peaked and tall atrial flutter waves in lead V_1 .

Case 127 Atrial Flutter with 4:1 Atrioventricular Block, Left Ventricular Hypertrophy and Possible Posterior Myocardial Infarction

The cardiac rhythm is atrial flutter with 4:1 A-V block (ventricular rate: 75 beats/min). The diagnosis of LVH is arrived at primarily by voltage criteria (see Case 11). In addition, posterior MI may be considered in view of the tall R wave in lead V_1 (see Case 65).

Case 128 Atrial Flutter with Wenckebach Atrioventricular Block and Aberrant Ventricular Conduction

The cardiac rhythm is atrial flutter with Wenckebach A-V block (the conduction ratios alternate between 2:1 and 4:1). Note that the long R-R interval is shorter than four atrial flutter cycles, whereas the short R-R interval is longer than two atrial flutter cycles, a characteristic feature of Wenckebach A-V block in atrial flutter (see Section 9). Every other QRS complex is slightly deformed because of the aberrant ventricular conduction due to Ashman's phenomenon (see Case 107). Lateral myocardial ischemia can be considered in view of the inverted T waves in lead V_5 .

Case 129 SSS manifested by Atrial Flutter with Far-Advanced Atrioventricular Block with Intermittent Atrioventricular Junctional Escape Rhythm Associated with Diaphragmatic Myocardial Infarction, Right Bundle Branch Block, and Low Voltage

The underlying cardiac rhythm is atrial flutter (atrial rate: 286 beats/min). The ventricular rate is extremely slow (rate: 20–45 beats/min) as a result of far-advanced A-V block. Some ventricular cycles are regular because A-V junctional escape rhythm (JER) occurs intermittently (ventricular rate: 45 beats/min). Other ECG abnormalities include RBBB and diaphragmatic MI (see Cases 19 and 65). The QRS amplitude in the limb leads is markedly reduced; this is called low voltage (see Case 57). This cardiac rhythm abnormality is a manifestation of advanced SSS (see Case 96).

Section 7

Atrioventricular Junctional Arrhythmias

Case 130 Atrioventricular Junctional Premature Contractions Causing Atrioventricular Junctional Bigeminy with Aberrant Ventricular Contraction

This ECG tracing was taken on a 65-year-old woman with palpitations. Arrows indicate ectopic P waves. The underlying cardiac rhythm is sinus (rate: 86 beats/min), and there are frequent atrioventricular junctional premature contractions (A-V JPCs; indicated by arrows) with aberrant ventricular conduction (AVC). Note that all A-V JPCs show bizarre QRS complexes (right bundle branch block pattern) due to AVC as a result of Ashman's phenomenon (see Case I07). A-V JPCs with AVC superficially mimic ventricular premature contractions (VPCs).

DIAGNOSTIC CRITERIA OF A-V JPC

- Premature retrograde P waves (inverted P waves in leads II, III, and aVF and upright P wave in lead aVR) may be preceded by or followed by QRS complexes in the presence of underlying sinus rhythm.
- The QRS complexes are normal unless there is AVC, right bundle branch block (RBBB), or left bundle branch block (LBBB).
- A constant coupling interval occurs.
- Premature retrograde P waves may not be followed by QRS complexes (rare).
- Premature normal (narrow) QRS complexes may not be preceded by or followed by retrograde P waves (not uncommon).

Case 131 Blocked Atrioventricular Junctional Premature Contractions Causing Atrioventricular Junctional Bigeminy with Aberrant Ventricular Contraction and Reciprocal Beats (Echo Beats)

This ECG tracing, obtained from a 75-year-old woman, was presented to the weekly ECG conference because of its interesting ECG findings. The underlying cardiac rhythm is sinus (marked S), but there are frequent A-V JPCs (indicating by arrows) producing A-V junctional bigeminy. It is interesting to note that many retrograde P waves are not followed by QRS complexes, meaning blocked A-V JPCs. In addition, some retrograde P waves are followed by QRS complexes (marked X) with long R-P intervals (called *reciprocal beats* or *echo beats*).

Furthermore, many QRS are slightly deformed because of AVC.

DIAGNOSTIC CRITERIA OF RECIPROCAL (ECHO) BEATS

- The premature P wave is conducted in a retrograde fashion (inverted P waves in leads II, III, and aVF, and upright P wave in lead aVR) after an A-V junctional beat or Wenckebach V-A or A-V block (at times after a marked first-degree A-V block).
- The premature retrograde P wave may or may not be followed by a QRS complex.
- The R-P interval is usually longer than 0.20 second.
- The R-R interval, including a sandwiched retrograde P wave, is usually 0.50 second or less (unless there is marked A-V or V-A conduction delay).

Case 132 Paroxysmal Atrioventricular Junctional Tachycardia with Left Ventricular Hypertrophy

The cardiac rhythm is paroxysmal A-V JT with a rate of 155 beats/min. Note that each QRS complex is preceded by a retrograde P wave, and the QRS complex is narrow (normal). It should be emphasized that a retrograde P wave may be preceded by or followed by a QRS complex in A-V JT, and at times, no P waves is discernible when the P wave is superimposed to the QRS complex. The diagnosis of left ventricular hypertrophy (LVH) is made using the conventional criteria (see Case 11).

Case 133 Nonparoxysmal Atrioventricular Junctional Tachycardia Associated with Acute Diaphragmatic Myocardial Infarction and Left Ventricular Hypertrophy

A hypertensive man was admitted to the coronary care unit because of an acute heart attack. The cardiac rhythm is nonparoxysmal A-V JT with a rate of 76 beats/min. Note that each retrograde P wave is followed by a QRS complex. The diagnosis of acute diaphragmatic myocardial infarction (MI) and LVH can be readily made using the conventional criteria (see Cases 1I and 65). Note that leads V_3 through V_5 are half-standardized.

Case 134 Nonparoxysmal Atrioventricular Junctional Tachycardia Associated with Acute Diaphragmatic Myocardial Infarction

These ECG rhythm strips were obtained from a patient with an acute heart attack. The cardiac

rhythm is nonparoxysmal A-V JT with a rate of 136 beats/min. There is no discernible P wave, and the QRS complex is narrow (normal). The diagnosis of acute diaphragmatic MI is established using the conventional criteria (see Case 65). It has been shown that nonparoxysmal A-V JT is most commonly due to acute diaphragmatic MI.

Case 135 Nonparoxysmal Atrioventricular Junctional Tachycardia Associated with Acute Pericarditis due to Trauma

This ECG tracing was taken on a 23-year-old woman following an automobile accident. The cardiac rhythm is nonparoxysmal A-V JT with a rate of 67 beats/min. Note that the ventricular cycle is slightly irregular (as is common in young individuals), but the QRS complex is normal with no P wave. Acute pericarditis can be diagnosed on the basis of the ST segment elevation diffusely in practically every ECG lead (see Section 12). The young woman developed acute pericarditis because of a steering wheel injury incurred in an auto accident.

Case 136 Nonparoxysmal Atrioventricular Junctional Tachycardia due to Digitalis Intoxication and Hypokalemia

These ECG rhythm strips were recorded from a patient with digitalis intoxication. The cardiac rhythm is nonparoxysmal A-V JT with a rate 76 beats/min. Note that QRS complex is followed by a retrograde P wave, and the QRS complex is normal. The diagnosis of hypokalemia is made on the basis of prominent U waves (see Case 10). Inexperienced readers may easily misinterpret as the prolonged QT interval. Digitalis intoxication is one of the most common causes of nonparoxysmal A-V JT, and hypokalemia is known to be a common predisposing factor for digitalis intoxication.

Case 137 Atrioventricular Junctional Tachycardia with 2:1 Atrioventricular Block Associated with Diffuse Subepicardial Injury, Low Voltage, and a Ventricular Premature Contraction

Arrows indicate retrograde P waves. The cardiac rhythm is A-V JT (atrial rate: 150 beats/min), with 2:1 A-V block and a VPC (in leads V_1 through V_3). Diffuse subepicardial injury involving diaphragmatic, posterior, and lateral wall is evident (see Case 59). The ST segment depression in leads V_1 through V_3 indicates a posterior subepicardial injury. The

QRS amplitude is significantly reduced in limb leads (low voltage) (see Case 57).

Case 138 Atrial Fibrillation with Nonparoxysmal Atrioventricular Junctional Tachycardia Producing Complete Atrioventricular Dissociation due to Digitalis Intoxication Associated with Hypokalemia

These cardiac rhythm strips were obtained from a patient with chronic congestive heart failure (CHF) and digitalis intoxication. The cardiac rhythm diagnosis is atrial fibrillation (AF) with nonparoxysmal A-V JT (ventricular rate: 92 beats/min) leading to complete A-V dissociation. Notice the prominent U waves (e.g., lead V₃) indicative of hypokalemia (see Case 10). As emphasized previously, digitalis intoxication most commonly produces nonparoxysmal A-V JT in the presence of the preexisting AF. In addition, hypokalemia commonly predisposes a patient to digitalis intoxication.

Case 139 Nonparoxysmal Atrioventricular Junctional Tachycardia with Incomplete Isorhythmic Atrioventricular Dissociation and Left Ventricular Hypertrophy

Arrows indicate the P waves of sinus origin. The underlying cardiac rhythm is sinus (atrial rate: 73 beats/min, indicated by arrows), but nonparoxysmal A-V JT (ventricular rate: 75 beats/min) occurs independently to control the ventricular activity, leading to incomplete isorhythmic A-V dissociation. The term *incomplete A-V dissociation* is used because there is a ventricular captured beat (i.e., normally conducted beat, the last beat in the rhythm strip lead II). Note that the atrial and the ventricular rates are almost identical, meaning they are isorhythmic. The diagnosis of LVH is made using the conventional criteria (see Case 11).

CAUSES OF A-V DISSOCIATION

- Slowing of sinus rate (sinus bradycardia)—sinus rate slower than A-V junctional (less commonly ventricular) escape rhythm
- Acceleration of A-V junctional (less commonly ventricular) impulse formation—A-V JT or ventricular tachycardia (VT)
- Advanced or complete A-V block
- Artificial pacemaker-induced ventricular rhythm
- Double (rarely triple) A-V junctional escape rhythms or tachycardias

Case 140 Atrial Flutter with Complete Atrioventricular Block and Nonparoxysmal Atrioventricular Junctional Tachycardia Producing Complete Atrioventricular Dissociation and a Ventricular Premature Contraction

Arrows indicate atrial flutter waves. There are two independent ectopic cardiac rhythms, including atrial flutter (indicated by arrows) and nonparoxysmal A-V JT (ventricular rate: 100 beats/min), leading to complete A-V dissociation. None of the atrial flutter waves are conducted to the ventricles, meaning there is a complete A-V block. As indicated earlier, complete A-V block is one of the common causes of complete A-V dissociation (see Case 139). In addition, there is a VPC (marked V).

Case 141 Atrial Tachycardia with Nonparoxysmal Atrioventricular Junctional Tachycardia Producing Incomplete Atrioventricular Dissociation Associated with Acute Diaphragmatic Myocardial Infarction and Acute Right Ventricular Myocardial Infarction

These ECG rhythm strips were recorded from a patient who had severe chest pain, 1 hour in duration. Arrows indicate P waves. The cardiac rhythm is atrial tachycardia (atrial rate: 157 beats/min) with nonparoxysmal A-V JT (rate: 72 beats/min), producing incomplete A-V dissociation. Note that the atrial and the ventricular activities are independent in most areas, but some QRS complexes (e.g., the 3rd and 10th beats) occur slightly earlier than the remaining QRS complexes. These prematurely occurring beats are the ventricular captured beats (the conducted atrial impulses). The diagnosis of acute diaphragmatic as well as the right ventricular M1 can be made using the conventional criteria (see Cases 65 and 81).

Case 142 Atrial Fibrillation and Atrioventricular Junctional Tachycardia with Wenckebach Exit Block Producing Complete Atrioventricular Dissociation due to Digitalis Intoxication

This ECG tracing was recorded from a patient with digitalis intoxication. The underlying cardiac rhythm is AF, but the ventricular cycle exhibits ventricular group beats followed by a ventricular pause. This ECG finding represents A-V JT with Wenckebach exit block of varying conduction ratios. Thus,

there is complete A-V dissociation. This type of cardiac arrhythmia is one of the most difficult ECG findings for most readers to understand. In order to understand the concept of Wenckebach exit block, every reader should study Wenckebach A-V block in the presence of sinus rhythm (see Section 9). Digitalis intoxication was responsible for production of this complex arrhythmia.

Case 143 Atrioventricular Junctional Escape Rhythm Associated with Hypokalemia

The cardiac rhythm is A-V junctional escape rhythm (JER) with a rate of 56 beats/min. Note that each normal QRS complex is followed by a retrograde P wave. The diagnosis of hypokalemia is made on the basis of prominent U waves (marked U) (see Case 10).

Case 144 Sinus Arrhythmia with Atrioventricular Junctional Escape Rhythm due to Complete Atrioventricular Block

The underlying cardiac rhythm is sinus arrhythmia (atrial rate: 60–82 beats/min), but an A-V junctional escape rhythm (ventricular rate: 36 beats/min) is present in the ventricles because of complete A-V block. In other words, as a result of complete A-V block, none of the sinus P waves are conducted to the ventricles. Thus, independently, the ventricles are activated by the A-V JER leading to complete A-V dissociation (see Case 139 and Section 9).

Case 145 Atrial Fibrillation with Atrioventricular Junctional Escape Rhythm due to Complete Atrioventricular Block

The underlying cardiac rhythm is AF, but the ventricular cycle is regular and slow. That is, the cardiac rhythm diagnosis is AF with A-V JER (ventricular rate: 47 beats/min) due to complete A-V block (see Section 9 and Case 139).

Case 146 Atrioventricular Junctional Escape Rhythm Associated with Anteroseptal Myocardial Infarction and Bifascicular Block Consisting of Right Bundle Branch Block and Left Anterior Hemiblock

Arrows indicate retrograde P waves. The cardiac rhythm is predominantly A-V JER (ventricular rate: 43 beats/min), and there is only one sinus beat (marked S). There are several ECG abnormalities

that include bifascicular block (BFB) consisting of RBBB and left anterior hemiblock (LAHB) associated with anteroseptal MI (see Cases 23 and 65). Note that each QRS complex is followed by a retrograde P wave (indicated by arrows) with constant R-P intervals. Sick sinus syndrome (SSS) is an underlying process in producing this cardiac arrhythmia (see Case 96).

Case 147 Sinus Arrhythmia with Intermittent Atrioventricular Junctional Escape Rhythm Producing Incomplete Atrioventricular Dissociation

Arrows indicate sinus P waves. The underlying cardiac rhythm is sinus arrhythmia (atrial rate: 55–62 beats/min), but A-V JER (ventricular rate: 56 beats/min) occurs intermittently, leading to incomplete A-V dissociation. In this case, A-V JER occurs intermittently when the sinus rate is reduced (see Case 139).

Case 148 Atrioventricular Junctional Escape Rhythm with Wenckebach Ventriculoatrial Block Associated with Reciprocal Beats and Aberrant Ventricular Conduction

Leads II-a, II-b, and II-c are continuous. The tracing exhibits A-V JER with retrograde Wenckebach ventriculoatrial block and frequent reciprocal beats occurring every third beat (marked RB). Note that the QRS complexes of all reciprocal beats (echo beats) are slightly deformed because of AVC (see Case 107). The diagnostic criteria of reciprocal beats were described earlier (see Case 131).

Case 149 Artificial Pacemaker—Induced Ventricular Rhythm with Wenckebach Ventriculoatrial Block and Reciprocal Beats

This ECG tracing was obtained from a patient with a permanent artificial pacemaker. The cardiac rhythm is artificial pacemaker-induced ventricular rhythm (ventricular rate: 73 beats/min) with Wenckebach ventriculoatrial (V-A) block and reciprocal beats (echo beats) occurring every third beat (see Section 9 and Case 131). A diagram of Wenckebach V-A block and reciprocal beats is provided (A, atrial; A-V, atrioventricular; V, ventricles). It is strongly advised that every reader study the Wenckebach A-V block first in order to understand Wenckebach V-A block (see Section 9).

Case 150 Double Atrioventricular Junctional Escape Rhythms Causing Atrioventricular Dissociation

Leads II-a to II-c are continuous. Downward arrows indicate retrograde P waves, whereas upward arrows indicate sinus P waves. The cardiac rhythm diagnosis is double A-V junctional escape rhythms leading to complete A-V dissociation. One of the A-V junctional pacemakers activates the atria in a retrograde fashion (indicated by downward arrows; atrial rate: 36 beats/min), whereas another A-V junctional pacemaker activates the ventricles (ventricular rate: 46 beats/min), independently leading to complete A-V dissociation (see Case 139). There are occasional sinus P waves (indicated by upward arrows) that are not conduced to the ventricles, judging from the regular ventricular cycle throughout (i.e., no disturbance of the ventricular cycle).

Case 151 Double Atrioventricular Junctional Escape Rhythms with a Reciprocal Beat Leading to Incomplete Atrioventricular Dissociation

Leads II-a, II-b, and II-c are continuous. The tracing shows double A-V JERs with a reciprocal beat (marked X) producing incomplete A-V dissociation (see Cases 131 and 139). One pacemaker in the A-V junction activates atria in a retrograde fashion (indicated by downward arrows), whereas another pacemaker in the A-V junction activates ventricles independently with similar rates, leading to incomplete A-V dissociation. Note the occasional sinus P waves (indicated by upward arrows) not conducted to the ventricles.

Case 152 Nonparoxysmal Atrioventricular Junctional Tachycardia with 3:2 Wenckebach Exit Block and Aberrant Ventricular Conduction Associated with Left Ventricular Hypertrophy and Anteroseptal Myocardial Infarction

This unusual arrhythmia belongs to a 67-year-old woman with known coronary artery disease (CAD), and this ECG tracing was presented to the weekly ECG conference. Clearly there is a regular irregularity of the ventricular cycle with no discernible P waves. The short and long R-R intervals alternate, and the longer R-R interval is shorter than two short R-R intervals. The cardiac rhythm diagnosis is nonparoxysmal A-V JT with 3:2 Wenckebach exit block. Some QRS complexes are slightly deformed because of AVC as a result of Ashman's phenomenon. Other ECG abnormalities include anterosep-

tal MI and LVH (see Cases 11 and 65). The exact nature of the atrial mechanism is uncertain, but AF is a good possibility. To understand the concept of Wenckebach exit block, every reader must study Wenckebach A-V block during sinus rhythm (see Section 9).

Section 8

Ventricular Arrhythmias

Case 153 Ventricular Premature Contractions with Ventricular Bigeminy

The underlying cardiac rhythm is sinus (rate: 88 beats/min), but there are frequent ventricular premature contractions (VPCs) producing ventricular bigeminy. Note that the VPCs and the sinus beats alternate on every other beat. The origin of the VPCs is the ventricular septum in view of the positive (upright) QRS complexes of all ectopic beats in leads V₁ as well as V₅. It is important to remember that the origin of the ectopic beats is always the ventricles whenever the ectopic beats are positive (upright) in all precordial leads.

Case 154 First-Degree Atrioventricular Block and Ventricular Premature Contractions with Ventricular Bigeminy Associated with Left Ventricular Hypertrophy

This ECG tracing was taken on a 72-year-old woman with chronic congestive heart failure (CHF) from long-standing hypertension. Arrows indicate sinus P waves. The underlying cardiac rhythm is sinus (rate: 100 beats/min), with first-degree A-V block; also there are frequent VPCs producing ventricular bigeminy. Inexperienced readers may misdiagnose this as APCs because the sinus P waves occur before VPCs; however, the P waves and VPCs are not related, of course. The diagnosis of LVH is made using the conventional criteria (see Case 11). In addition, a remote possibility of diaphragmatic MI should be considered.

Case 155 Interpolated Ventricular Premature Contractions with Ventricular Bigeminy

The underlying cardiac rhythm is sinus bradycardia (rate: 58 beats/min), but there are frequent interpolated VPCs producing areas of ventricular bigeminy. This ECG is within normal limits, otherwise.

Case 156 Atrial Fibrillation with Atrioventricular Junctional Escape Rhythm due to Complete Atrioventricular Block and Ventricular Premature Contractions with Ventricular Bigeminy due to Digitalis Intoxication

These ECG rhythm strips were obtained from an elderly patient with digitalis intoxication. Leads II-a and II-b, and V_{1-a} and V_{1-b} are continuous in each given lead. The underlying cardiac rhythm is AF with A-V junctional escape rhythm (indicate by arrows) due to complete A-V block (see Section 9). There are frequent VPCs (marked X) producing ventricular bigeminy. These cardiac arrhythmias are usually a manifestation of advanced digitalis intoxication.

Case 157 Ventricular Premature Contractions with Reciprocal Beats (Echo Beats) Associated with Diaphragmatic Myocardial Infarction

This ECG tracing was taken on a patient who had had a recent heart attack. The underlying cardiac rhythm is sinus bradycardia (rate: 50 beats/min), with VPCs followed by consecutively occurring reciprocal beats (echo beats). Reciprocal beats are the result of a reentry phenomenon in the A-V junction (see Case 131). The diagnosis of recent diaphragmatic MI can be established using the conventional criteria (see Case 65).

Case 158 Ventricular Premature Contractions with Ventricular Group Beats Leading to Nonsustained Ventricular Tachycardia

All cardiac rhythm strips of lead II are continuous. The underlying cardiac rhythm is sinus (rate: 75–105 beats/min), but there are frequent VPCs (marked X) with ventricular group beats and several episodes of nonsustained ventricular tachycardia (VT) with a rate of 215 beats/min. VT is said to be nonsustained when it lasts 29 seconds or less; VT is sustained when it lasts more than 29 seconds.

ECG FINDINGS CAUSING BROAD QRS COMPLEXES

- Ventricular ectopies
- Intraventricular block
- Aberrant ventricular conduction
- Wolff-Parkinson-White (WPW) syndrome
- Artificial pacemaker–induced ventricular beats or rhythm

ECG FINDINGS SUPPORTIVE OF (OR DIAGNOSTIC OF) VENTRICULAR ECTOPIES

- Full compensatory pause during sinus rhythm (occasionally interpolated)—no disturbance on the sinus P-P cycle
- Significant pause after a bizarre QRS complex in atrial fibrillation or flutter
- No premature P wave preceding a bizarre QRS complex
- Extremely broad and bizarre QRS complex without showing typical left bundle branch block (LBBB) or right bundle branch block (RBBB) pattern
- Bizarre beat showing a positive (upright) QRS complex in all precordial leads
- Occurrence of ventricular fusion beat(s) during consecutively occurring bizarre beats (e.g., ventricular group beats or VT)
- No evidence of Ashman's phenomenon
- No response to carotid sinus stimulation (CSS) during consecutively occurring bizarre beats (e.g., VT)

A-V dissociation usually, but not always, favors VT.

Case 159 Ventricular Premature Contractions and Sustained Ventricular Tachycardia Associated with Acute Diaphragmatic Myocardial Infarction

The ECG tracings A and B were taken on a patient with recent M1. The underlying cardiac rhythm is sinus, but there are VPCs (tracing B) and sustained VT (rate: 187 beats/min) in tracing A. Note that the configuration of VPCs and VT complexes are identical, which means that the same ectopic focus produces VPCs as well as VT. The diagnosis of recent diaphragmatic MI (pathologic Q waves in leads 11, III, and aVF in 12-lead ECG—not shown here) is made using the conventional criteria (see Case 65). Various ECG findings diagnostic of (or supportive of) ventricular ectopies were summarized previously (see Case 158).

Case 160 Sustained Ventricular Tachycardia

This ECG tracing was recorded from a 53-year-old woman with cardiomyopathy. The atrial activities are not discernable, and the QRS complexes are broad with a regular ventricular cycle. The cardiac rhythm diagnosis is sustained VT with a rate of 152 beats/min. In this case, VT is considered to be originating from the left ventricle, judging from the positive (upright) QRS complex in lead V₁ with the negative QRS complexes in leads I, aVL, and V₆.

Various ECG findings which are diagnostic of (or supportive of) ventricular ectopies were described earlier (see Case 158). It is important to recognize that the configuration of the bizarre QRS complexes during VT fails to show a typical LBBB or RBBB pattern.

Case 161 Sustained Ventricular Tachycardia

This ECG tracing was taken on a 57-year-old woman with known coronary artery disease (CAD). The atrial activities are not discernable, and QRS complexes are broad with a regular ventricular cycle. The cardiac rhythm diagnosis is a relatively slow, sustained VT with a rate of 120 beats/min. Various ECG findings that are diagnostic of (or supportive of) ventricular ectopies were summarized previously (see Case I58). It should be emphasized that the configuration of the bizarre QRS complexes during VT fails to reveal typical LBBB or RBBB pattern.

Case 162 Bidirectional Nonsustained Ventricular Tachycardia and Interpolated Ventricular Premature Contractions

The underlying cardiac rhythm is sinus bradycardia (rate: 48 beats/min), but there are frequent interpolated VPCs (marked V), and bidirectional nonsustained VT (rate: 185 beats/min). Leads Il-a to Il-c are not continuous.

Case 163 Atrial Fibrillation with Bidirectional Sustained Ventricular Tachycardia Leading to Complete Atrioventricular Dissociation due to Digitalis Intoxication

This ECG tracing was obtained from a 70-year-old man with chronic obstructive pulmonary disease (COPD) and thyrotoxicosis; he expired soon after this ECG was recorded. Digitalis intoxication was confirmed by his serum digoxin level determination (more than 10 ng/mL). The cardiac rhythm diagnosis is AF with sustained bidirectional VT (rate: 170 beats/min), producing complete A-V dissociation (see Case 139).

Case 164 Atrial Tachycardia with Sustained Bidirectional Ventricular Tachycardia Producing Complete Atrioventricular Dissociation

Arrows indicate P waves, and leads II-a and II-b, and leads V_{1-a} and V_{1-b} are continuous in each given lead. The cardiac rhythm diagnosis is atrial tachycardia (atrial rate: 186 beats/min) with sustained bidirectional VT (rate: 153 beats/min) producing

complete A-V dissociation (see Cases 139 and 158). Digitalis intoxication is responsible for the production of these arrhythmias.

Case 165 Sick Sinus Syndrome Manifested by Brady-Tachyarrhythmia Syndrome Consisting of Atrial Flutter-Fibrillation with Intermittent Atrioventricular Junctional Escape Rhythm due to Advanced Atrioventricular Block and Nonsustained Ventricular Tachycardia Associated with Hypokalemia

The underlying cardiac rhythm is atrial flutter-fibrillation with advanced A-V block and intermittent A-V JER (ventricular rate: 38 beats/min). Two episodes of nonsustained VT (rate: 150 beats/min) occur in the presence of atrial flutter-fibrillation leading to brady-tachyarrhythmia syndrome (BTS), which is a form of advanced SSS (see Cases 96 and 158). Hypokalemia is considered because of prominent U waves (see Case 10).

Case 166 Torsade de Pointes due to Quinidine Toxicity

These ECG rhythm strips were recorded from a patient with quinidine toxicity. Leads II-a to II-e are continuous. The underlying cardiac rhythm is sinus (rate: 65 beats/min), with first-degree A-V block. Multiformed VT is initiated by frequent VPCs (marked V) with the R-on-T phenomenon due to the markedly prolonged QT interval with broad T waves that result from quinidine toxicity. Multiformed VT has transformed to ventricular fibrillation (VF). This ventricular tachyarrhythmia is termed torsade de pointes. Torsade de pointes is the most serious complication of quinidine (and less commonly procainamide) toxicity.

Case 167 Ventricular Flutter

This ECG tracing was recorded form a patient with acute MI. The cardiac rhythm diagnosis is ventricular flutter with a rate of 210 beats/min. In ventricular flutter, there is no boundary between QRS complexes, ST segments, and T waves, so the entire ECG complex appears to be a continuous loop formation. The clinical significance of ventricular flutter is almost the same as that of VF.

Case 168 Ventricular Fibrillation

These cardiac rhythm strips were taken on a patient during the cardiac arrest as a result of acute M1. Leads II-a to II-c are continuous. The cardiac rhythm diagnosis is VF.

Case 169 Sinus Arrhythmia with Intermittent Sinus Arrest and Ventricular Escape Beats Producing Incomplete Atrioventricular Dissociation Associated with Left Ventricular Hypertrophy

The underlying cardiac rhythm is sinus arrhythmia with sinus bradycardia (rate: 55–73 beats/min). Ventricular escape beats occur intermittently when the sinus node fails to produce the cardiac impulses (sinus arrest), leading to incomplete A-V dissociation (see Cases 96, 139, and 158). There are occasional ventricular fusion beats (e.g., the third QRS complex in the rhythm strip lead II). The diagnosis of LVH is established using the conventional criteria (see Case 11).

Case 170 Sinus Rhythm with Wenckebach Atrioventricular Block and Ventricular Escape Beats Producing Incomplete Atrioventricular Dissociation Associated with Diaphragmatic and Anteroseptal Myocardial Infarction

This ECG tracing was taken on a 64-year-old man with CAD. Arrows indicate P waves. The cardiac rhythm diagnosis is sinus rhythm (indicated by arrows; atrial rate: 70 beats/min), with slowly progressing Wenckebach A-V block and intermittent VER (marked V; ventricular rate: 37 beats/min) producing incomplete A-V dissociation (see Section 9 and Cases 139 and 158). The diagnosis of diaphragmatic MI is established using the conventional criteria (see Case 65), and a possibility of anteroseptal MI is present. In addition, LVH is diagnosed (see Case 11).

Case 171 Sinus Rhythm with Ventricular Escape Rhythm due to Complete Atrioventricular Block

Arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus rhythm (indicated by arrows; atrial rate: 93 beats/min) with ventricular escape rhythm (VER; ventricular rate: 32 beats/min) due to complete A-V block (see Section 9). Note that the atrial and ventricular activities are independent throughout because none of the atrial impulses are conducted to the ventricles as a result of complete A-V block, leading to complete A-V dissociation. It should be emphasized that complete A-V block is one of the common causes of complete A-V dissociation (see Case 139).

Case 172 Ventricular Dissociation Consisting of Ventricular Escape Rhythm and Independent Unilateral Ventricular Flutter

Leads II-a to II-d are continuous. The underlying cardiac rhythm is a slow ventricular escape rhythm (VER) with a rate of 23 beats/min. It is interesting to note intermittent unilateral ventricular flutter leading to ventricular dissociation. The term unilateral ventricular flutter is used because a slow and regular VER is not disturbed by ventricular flutter leading to ventricular dissociation. The board term chaotic ventricular rhythm is used to describe a variety of unstable ventricular arrhythmias such as this one.

Section 9

Atrioventricular Block

Case 173 Sinus Rhythm with First-Degree
Atrioventricular Block due to Acute
Diaphragmatic Myocardial Infarction
Associated with Posterior
Subepicardial Injury and
Possible Old Anteroseptal
Myocardial Infarction

This ECG tracing was taken on an 80-year-old woman with an acute heart attack. The cardiac rhythm is sinus (rate: 75 beats/min) with first-degree atrioventricular (A-V) block (P-R interval: 0.26 second). The diagnosis of acute diaphragmatic myocardial infarction (MI) with posterior subepicardial injury is made using the conventional criteria (see Case 65). In addition, a possibility of old anteroseptal MI is strongly considered.

CLASSIFICATION OF A-V BLOCK (ACCORDING TO THE DEGREE OF THE BLOCK)

- First-degree A-V block
- Second-degree A-V block
 - Wenckebach (Mobitz type I) A-V block
 - Mobitz type II A-V block
 - -2:I A-V block
- Advanced (high degree) A-V block
- Complete (third-degree) A-V block

CLASSIFICATION OF A-V BLOCK (ACCORDING TO THE SITE OF THE BLOCK)

- A-V nodal block (intranodal block)
- Infranodal block
 - Intra-His block
 - Infra-His block

Case 174 Sinus Rhythm with Wenckebach Atrioventricular Block Associated with Ventriculophasic Sinus Arrhythmia and Anterior Myocardial Ischemia and Left Ventricular Hypertrophy

This ECG tracing was obtained from a patient with coronary artery disease (CAD) and hypertension. Arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus rhythm (rate: 72 beats/min) with 3:2 Wenckebach A-V block. It is noteworthy that the P-P interval, including a QRS complex, is shorter than the P-P interval without QRS complex during Wenckebach A-V block. This finding is termed *ventriculophasic sinus arrhythmia*. Other ECG abnormalities include left ventricular hypertrophy (LVH) and diffuse anterior ischemia (see Cases 11 and 55).

DIAGNOSTIC CRITERIA OF WENCKEBACH A-V BLOCK

- The P-R intervals progressively lengthen until a blocked P wave occurs.
- There is progressive shortening of the R-R intervals (ventricular cycles) until a blocked P wave occurs.
- The longest R-R interval is shorter than two P-P cycles.
- The A-V conduction ratios may be constant or variable.
- Regular irregularity of the R-R intervals (ventricular cycles) occurs when the A-V conduction ratio is fixed (constant).

Case 175 Sinus Rhythm with 4:3 Wenckebach Atrioventricular Block

Leads II-a and II-b are continuous. Arrows indicate P waves. The cardiac rhythm diagnosis is sinus rhythm (atrial rate: I00 beats/min) with 4:3 Wenckebach A-V block (see Case 174). The P waves are somewhat peaked and the finding is suggestive of right atrial enlargement (RAE) (see Case 8).

Case 176 Sinus Rhythm with Wenckebach Atrioventricular Block and Atrioventricular Junctional Escape Beats Producing Incomplete Atrioventricular Dissociation

Arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus rhythm (atrial rate: 64 beats/min) with 3:2 and 4:3 Wenckebach A-V block and occasional A-V junctional escape beats leading to incomplete A-V dissociation (see Cases I39 and 174).

Wenckebach A-V block is best shown in the last section of the rhythm strip lead II.

Case 177 Sinus Tachycardia with 2:1 Atrioventricular Block Associated with Acute Diaphragmatic—Lateral Myocardial Infarction

This ECG tracing was taken on a 63-year-old woman with an acute heart attack. Arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus tachycardia (atrial rate: 115 beats/min) with 2:1 A-V block. In this case, 2:1 A-V block represents a variant of Wenckebach A-V block, judging from normal QRS complexes in all conducted beats associated with acute diaphragmatic MI (see Cases 65, 173, and 174). The diagnosis of acute diaphragmatic-lateral MI is readily established using the conventional criteria (see Case 65). In addition, the slight ST segment depression in leads V_1 through V_2 represents posterior subepicardial injury, which often occurs with acute diaphragmatic MI (see Case 59).

Case 178 Sinus Rhythm with 2:1 Atrioventricular Block Associated with Acute Diaphragmatic Myocardial Infarction and Old Anteroseptal Myocardial Infarction

This ECG tracing was recorded in the emergency room on an 80-year-old woman with severe chest pain of 1 to 2 hours' duration. Arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus rhythm (atrial rate: 78 beats/min) with 2:1 A-V block, which is considered to be a variant of Wenckebach A-V block (see Cases 173 and 174). Some inexperienced readers may not recognize every other blocked P wave, because of their superimposition to the end portion of the T waves of the preceding beats. Other ECG abnormalities include acute diaphragmatic MI, old anteroseptal MI and left atrial enlargement (LAE) (see Cases 6 and 65). In addition, diffuse anterior subendocardial injury is considered (see Case 63).

Case 179 Sinus Rhythm with 3:2 Wenckebach Sinoatrial Block and 3:2 Wenckebach Atrioventricular Block

Arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus rhythm (atrial rate: 73 beats/min) with 3:2 Wenckebach S-A block associated with 3:2 Wenckebach A-V block (see Cases 100 and 174). By and large, Wenckebach S-A block is one of the most difficult arrhythmias to diagnose for

most readers. Every reader should understand Wenckebach A-V block before interpreting Wenckebach S-A block. It should be noted that the long P-P interval is shorter than two short P-P intervals—a characteristic feature of Wenckebach S-A block. In addition, the periodic occurrence of the expected blocked sinus P waves fails to appear during Wenckebach A-V block because of a coexisting Wenckebach S-A block.

Case 180 Sinus Rhythm with 3:1 Advanced Atrioventricular Block

Leads II-a and 11-b are continuous. The cardiac rhythm diagnosis is sinus (atrial rate: 64 beats/min) with 3:1 advanced A-V block (see Case 173). In general, 3:1 A-V block is an extremely uncommon finding in our daily practice. Arrows indicate sinus P waves.

Case 181 Sinus Rhythm with 3:1 Advanced Atrioventricular Block and Ventricular Escape Beats Leading to Ventricular Escape-Bigeminy Associated with Left Ventricular Hypertrophy by Voltage

Arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus rhythm (atrial rate: 64 beats/min) with 3:1 advanced A-V block and frequent ventricular escape beats (marked X) producing ventricular escape-bigeminy (see Cases 158 and 173). The diagnosis of LVH is considered by voltage criteria alone. It should be noted that the QRS complexes of the ventricular escape beats (marked X) are only slightly deformed.

Case 182 Sinus Rhythm with Wenckebach Atrioventricular Block and Intermittent Demand Ventricular Pacemaker Beats Associated with Diffuse Myocardial Ischemia and Left Ventricular Hypertrophy

Arrows indicate sinus P waves. The underlying cardiac rhythm is sinus (atrial rate: 88 beats/min) with 3:2 Wenckebach A-V block (see Case 174), and there are frequent demand ventricular pacemaker beats (marked X). Many pacing beats exhibit a different QRS configuration because of ventricular fusion beats of varying degrees. Other ECG abnormalities include diffuse myocardial ischemia and LVH (see Cases 11 and 55).

Case 183 Mobitz Type II Atrioventricular Block with Bifascicular Block Consisting of Right Bundle Branch Block and Left Anterior Hemiblock Leading to Incomplete Trifascicular Block Associated with Posterolateral Myocardial Infarction

This ECG tracing was taken on a patient with known CAD. Arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus rhythm (atrial rate: 62 beats/min) with Mobitz type II A-V block. Note that all conducted sinus beats show constant P-R intervals; blocked sinus P waves (marked X) occur intermittently. Other ECG abnormalities include bifascicular block (BFB) consisting of right bundle branch block (RBBB) and left anterior hemiblock (LAHB) associated with posterolateral MI (see Cases 23 and 65). Incomplete trifascicular block (TFB) is diagnosed on the basis of a combination of Mobitz type II A-V block and BFB (see Case 23). Permanent artificial pacemaker implantation is highly recommended in this patient.

DIAGNOSTIC CRITERIA OF MOBITZ TYPE II A-V BLOCK

- Periodic or intermittent occurrence of blocked P waves without Wenckebach phenomenon
- Constant P-R intervals in all conducted beats
- An R-R interval, including the blocked P wave, is two times the P-P interval
- Regular irregularity of the R-R cycles when the A-V conduction ratio is constant
- Common association with hemiblocks, left bundle branch block (LBBB), RBBB, or BFB

Case 184 Advanced Mobitz Type-II Atrioventricular Block with Left Anterior Hemiblock and Intermittent Ventricular Escape Rhythm Leading to Incomplete Atrioventricular Dissociation

Leads II-a to II-d are continuous, and arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus rhythm (atrial rate: 72 beats/min) with advanced Mobitz type II A-V block (see Case 183) leading to intermittent ventricular escape rhythm (VER) (ventricular rate: 35 beats/min). As a result, incomplete A-V dissociation is produced (see Case 139). The other ECG abnormality is, obviously, LAHB (see Case 36). At a glance, some portion of lead II-d appears to show sinus rhythm with 2:1 A-V block, but the atrial and the ventricular activities are independent, except for the first beat in lead II-d. Remember that all QRS complexes of the

VER show the identical configuration (leads II-a and II-d).

Case 185 Sinus Rhythm with 2:1 Atrioventricular Block (Variant of Mobitz Type II Atrioventricular Block) Associated with Bifascicular Block Consisting of Right Bundle Branch Block and Left Anterior Hemiblock Leading to Incomplete (Partial) Trifascicular Block

The cardiac rhythm diagnosis is sinus rhythm (atrial rate: 74 beats/min) with 2:1 A-V block. Other ECG abnormalities include BFB consisting of RBBB and LAHB. In this case, 2:1 A-V block is considered to be a variant of Mobitz type II A-V block because of coexisting BFB. A combination of 2:1 A-V block and BFB is strongly suggestive of incomplete (partial) TFB (see Cases 23 and 183).

Case 186 Atrial Tachycardia with Wenckebach Atrioventricular Block due to Digitalis Intoxication

Arrows indicate ectopic P waves, and leads V_{1-a} to V_{1-c} are continuous. The cardiac rhythm diagnosis is atrial tachycardia (atrial rate 214 beats/min) with Wenckebach A-V block (see Case 174 and Section 6). Atrial tachycardia with Wenckebach A-V block is often called "paroxysmal atrial tachycardia (PAT) with block," and digitalis intoxication is the most common underlying cause of this arrhythmia.

Case 187 Atrial Flutter with Wenckebach Atrioventricular Block Associated with Lateral Myocardial Ischemia

The cardiac rhythm reveals atrial flutter (atrial rate: 286 beats/min) with Wenckebach A-V block of varying A-V conduction ratios. Note that grouped QRS complexes (2 to 5 beats in a row) are followed by a pause, and this ECG finding is a characteristic feature of Wenckebach A-V block (see Case 174 and Section 6). In addition, lateral myocardial ischemia is considered in view of inverted T wave in lead V₅.

Case 188 Atrial Flutter with 4:1 Atrioventricular Block Associated with Left Ventricular Hypertrophy and Hypokalemia

This ECG tracing was obtained from an 84-year-old woman with chronic congestive heart failure (CHF) from long-standing hypertension. The cardiac rhythm is atrial flutter (atrial rate: 212 beats/min) with 4:1 A-V block (ventricular rate: 53 beats/min) (see Section 6). The atrial flutter cycle is much

slower than usual because of the quinidine effect. Other ECG abnormalities include LVH and prominent U waves indicative of hypokalemia (see Cases 10 and 11).

Case 189 Sinus Rhythm with Atrioventricular Junctional Escape Rhythm due to Advanced Atrioventricular Block Associated with Acute Diaphragmatic Myocardial Infarction and Left Ventricular Hypertrophy

These ECG rhythm strips were obtained from a patient with acute heart attack. The cardiac rhythm is sinus (atrial rate: 78 beats/min) with predominantly A-V junctional escape rhythm (JER) (ventricular rate: 43 beats/min) due to advanced A-V block leading to incomplete A-V dissociation (see Cases 139 and 173). Note a ventricular captured beat (normally conducted sinus beat, the last beat). The diagnosis of acute diaphragmatic MI and LVH is obvious using the conventional criteria (see Cases 11 and 65).

Case 190 Sinus Rhythm with Atrioventricular Junctional Escape Rhythm due to Complete Atrioventricular Block Associated with Left Ventricular Hypertrophy

Arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus rhythm (atrial rate: 77 beats/min) with A-V JER (ventricular rate: 38 beats/min) due to complete A-V block (see Case 173). Note that the atrial and the ventricular activities are independent throughout, leading to compete A-V dissociation. The diagnosis of LVH is made using the conventional criteria (see Case 11).

Case 191 Sinus Rhythm with Ventricular Escape Rhythm due to Complete Atrioventricular Block

This ECG tracing was taken on a 77-year-old man with frequent fainting episodes. Arrows indicate sinus P waves. The cardiac rhythm diagnosis is sinus rhythm (atrial rate: 87 beats/min) with VER (ventricular rate: 43 beats/min) due to complete A-V block (see Cases 158 and 173). Note that the QRS complexes are broad and bizarre with a regular and slow ventricular cycle.

Case 192 Atrial Fibrillation with Atrioventricular Junctional Escape Rhythm due to Complete Atrioventricular Block

The underlying cardiac rhythm is atrial fibrillation (AF), but the ventricular activity is independent to

the atrial activity. Namely, the cardiac rhythm diagnosis is AF with A-V JER (ventricular rate: 38 beats/min) due to complete A-V block (see Case 173). This cardiac arrhythmia strongly suggests advanced sick sinus syndrome (SSS) (see Case 96).

Case 193 Atrial Flutter with Atrioventricular Junctional Escape Rhythm due to Complete Atrioventricular Block

The cardiac rhythm diagnosis is atrial flutter (atrial rate: 286 beats/min) with A-V JER (ventricular rate: 48 beats/min) due to complete A-V block (see Case 173). Inexperienced readers may misdiagnose as atrial flutter with 5:1 or 6:1 A-V block simply by calculating the atrial to the ventricular activities. However, it should be emphasized that the atrial and the ventricular activities are independent throughout, leading to complete A-V dissociation (see Case 139).

Section 10

Wolff-Parkinson-White Syndrome (Ventricular Preexcitation Syndrome)

Case 194 Wolff-Parkinson-White Syndrome, Type B

This ECG tracing was taken on a 29-year-old apparently healthy man. The cardiac rhythm is sinus bradycardia with arrhythmia (rate: 50–54 beats/min). The ECG diagnosis is Wolff-Parkinson-White (WPW) syndrome, type B. Notice the short PR interval with a broad QRS complex due to a delta wave. Pseudo diaphragmatic myocardial infarction (MI) pattern is produced because the delta wave is directed inferiorly (see Case 84).

WPW syndrome is a unique congenital anomaly, and people with WPW syndrome are born with one or more bypass tracts connecting between the atria and the ventricles. The bypass tract is responsible for a unique ECG finding that consists of a short PR interval and a broad QRS complex. Other congenital anomalies (e.g., atrial septal defect, Ebstein's anomaly) often coexist with WPW syndrome.

DIAGNOSIS CRITERIA OF WPW SYNDROME, TYPE B

- The short PR interval (usually less than 0.12 second) with a broad QRS complex (usually more than 0.10 second) is due to a delta wave (initial slurring of the QRS complex).
- The delta wave is directed posteriorly and either inferiorly or superiorly.
- The QRS complexes in leads V_1 through V_3 (at times only leads V_1 or V_1 through V_2) disclose QS waves or rS complexes, whereas leads V_4 through V_6 show tall and broad QRS complexes.

- A pseudo MI pattern is often produced (diaphragmatic or anteroseptal).
- Various supraventricular tachyarrhythmias often occur.
- Secondary ST segment and T wave changes occur in leads I, aVL, and V₄ through V₆.

Case 195 Wolff-Parkinson-White Syndrome, Type A

This ECG tracing belongs to a 19-year-old apparently healthy man. The cardiac rhythm is sinus bradycardia, with a rate of 54 beats/min. The ECG diagnosis is WPW syndrome, type A. It is important to remember that WPW syndrome is the major diagnostic possibility when the QRS complexes are upright (positive) in all precordial leads.

DIAGNOSTIC CRITERIA OF WPW SYNDROME, TYPE A

- The short PR interval (usually less than 0.12 second) with a broad QRS complex (usually more than 0.10 second) is due to a delta wave.
- The delta wave is directed anteriorly and either inferiorly or superiorly.
- The QRS complexes are often upright (positive) in all precordial leads.
- Pseudo MI pattern is often produced (diaphragmatic and posterior).
- Secondary ST segment and T wave changes occur in various ECG leads.
- Various supraventricular tachyarrhythmias often occur.

Case 196 Intermittent Wolff-Parkinson-White Syndrome

The cardiac rhythm is sinus arrhythmia, with a rate ranging from 70 to 93 beats/min. There are two kinds of QRS complexes because of intermittent WPW syndrome (marked X). In other words, the sinus impulses may be conducted to the ventricles via an accessory pathway intermittently (marked X) and the finding is rate-independent. Intermittent WPW syndrome superficially mimics ventricular premature contractions (VPCs) with group beats. A pseudo diaphragmatic MI pattern is produced because the delta wave is directed inferiorly (see Case 84).

Case 197 Intermittent Wolff-Parkinson-White Syndrome on Every Other Beat Associated with Left Anterior Hemiblock and Left Ventricular Hypertrophy

This ECG tracing exhibits sinus rhythm (rate: 100 beats/min) and intermittent WPW syndrome that

occurs on every other beat. In other words, the sinus impulses are conducted to the ventricles via the normal atrioventricular (A-V) conduction system and an accessory pathway (bypass tract) alternately. This ECG finding superficially mimics VPCs with ventricular bigeminy. Other ECG findings include left anterior hemiblock (LAHB) and left ventricular hypertrophy (LVH) (see Cases I1 and 36).

Case 198 Wolff-Parkinson-White Syndrome with Multiple Accessory Pathways (Bypass Tracts)

This Holter monitor ECG was recorded on a 24-year-old man with repetitive episodes of palpitations. The ECG rhythm strips A to D are not continuous. The cardiac rhythm is marked sinus arrhythmia, with sinus bradycardia (rate: 42–70 beats/min). There are four kinds of QRS complexes, and all of them show delta waves (different degrees). This ECG finding demonstrates WPW syndrome with multiple accessory pathways (bypass tracts). In other words, this patient was born with multiple bypass tracts and all of them function on different occasions (see Cases 194 and 195).

Case 199 Reciprocating (Reentrant) Tachycardia in Wolff-Parkinson-White Syndrome

This ECG tracing was recorded during one episode of palpitations. The QRS complexes are normal (narrow) with no discernible P waves, and the ventricular cycle is precisely regular. The cardiac rhythm diagnosis is reciprocating tachycardia (rate: 148 beats/min) due to WPW syndrome. In this case, the cardiac impulses are conducted to the ventricles via the normal A-V conduction system, and the impulses activate the atria via a bypass tract in a retrograde fashion.

Reciprocating tachycardia with normal QRS complexes is the most common tachyarrhythmia associated with WPW syndrome. Intravenous adenosine is extremely effective to terminate this type of tachycardia.

Case 200 Atrial Fibrillation with Anomalous Conduction (Conduction via a Bypass Tract) Associated with Wolff-Parkinson-White Syndrome, Type A

The ECG tracings shown in Cases 200 and 201 were obtained from a 24-year-old man with many episodes of palpitations. The cardiac rhythm is atrial fibrillation (AF) with extremely rapid ventricular rate (rate: 240–300 beats/min) and markedly bizarre QRS complexes. Namely, the ECG diagno-

sis is AF with anomalous A-V conduction (conduction via accessory pathway) due to WPW syndrome, type A (see Cases 195 and 201). Ventricular tachycardia is closely simulated, but the grossly irregular ventricular cycle excludes such a possibility.

For the treatment, various antiarrhythmic agents such as quinidine, procainamide, and amiodarone may be tried. If the drug therapy is ineffective, various surgical approaches or catheter ablation should be considered. For urgent treatment, DC shock should be applied immediately. It is extremely important to remember that digitalis or verapamil is contraindicated in the treatment of various tachyarrhythmias with broad QRS complexes in WPW syndrome because the conduction via a bypass tract is often accelerated, leading to more serious life-threatening tachyarrhythmias (e.g., ventricular fibrillation) and even death.

Case 201 Wolff-Parkinson-White Syndrome, Type A

The ECG tracings shown in Cases 200 and 201 were obtained from a 24-year-old man with many episodes of palpitations. The cardiac rhythm is marked sinus arrhythmia (rate: 52–63 beats/min), and the diagnosis of WPW syndrome, type A is obvious using the conventional criteria (see Case 195). Pseudo diaphragmatic-posterior MI pattern is produced because the delta wave is directed inferiorly and anteriorly (see Case 84).

Case 202 Atrial Fibrillation with Anomalous Conduction (Conduction via Bypass Tract) in Wolff-Parkinson-White Syndrome, Type B

This ECG tracing was taken on a 22-year-old man with frequent episodes of palpitations. The ECG diagnosis is AF with an extremely rapid ventricular response (rate: 200–260 beats/min) and anomalous A-V conduction (conduction via accessory pathway) due to WPW syndrome, type B. Note that some QRS complexes are more bizarre because of aberrant ventricular conduction due to extremely rapid ventricular rate in addition to anomalous A-V conduction itself (see Case 107). Ventricular tachycardia is closely simulated.

Case 203 Wolff-Parkinson-White Syndrome, Type B, Associated with Left Bundle Branch Block

This ECG tracing was recorded from a 66-year-old man with a history of rapid heart action since childhood. The cardiac rhythm is markedly slow sinus bradycardia (rate: 43 beats/min). The QRS com-

plexes are very broad and bizarre because of two coexisting ECG abnormalities including left bundle branch block (LBBB) and WPW syndrome, type B (see Cases 26, 158, and 194).

Case 204 Wolff-Parkinson-White Syndrome, Type B, Associated with Left Bundle Branch Block and Left Atrial Bradycardia

This ECG tracing was obtained from a 66-year-old man with repetitive episodes of palpitations. Arrows indicate P waves. The cardiac rhythm is left atrial bradycardia (rate: 53 beats/min). Note that the ectopic P waves are inverted in leads II, III, and aVF, and lead V₁ shows only upright component of P waves meaning that the ectopic impulses arise from left atrium (see Section I1). The QRS complexes are extremely broad and bizarre because of two coexisting ECG abnormalities: LBBB and WPW syndrome, type B (see Cases 26 and 194).

Case 205 Wolff-Parkinson-White Syndrome, Type B, Associated with Left Bundle Branch Block and Intermittent Sinoatrial Block Leading to Ventricular Escape Beats

This complicated ECG tracing was recorded from a 66-year-old man with multiple complaints that included palpitations. Arrows indicate sinus P waves. The underlying cardiac rhythm is sinus bradycardia (rate: 54 beats/min), but there is a period of 3:1 sinoatrial (S-A) block (in the first half of the rhythm strip lead II). Note that a long P-P interval is three times the basic P-P cycle, meaning 3:1 type II S-A block (see Case 99). During S-A block, ventricular escape beats (marked E) occur intermittently, leading to incomplete A-V dissociation (see Cases 139 and 158). Also notice the occasional VPCs (marked V). Other major ECG abnormalities include WPW syndrome type B, and LBBB (see Cases 26 and 194). Sick sinus syndrome (SSS) should be considered as a cause of the S-A block (see Case 96).

Case 206 Atrial Flutter with 2:1 Atrioventricular Conduction and Ventricular Premature Contractions Associated with Wolff-ParkinsonWhite Syndrome, Type A, and Right Bundle Branch Block

This ECG tracing was taken on a 70-year-old man with frequent episodes of palpitation. The underlying cardiac rhythm is atrial flutter (atrial rate: 225 beats/min) with 2: I A-V conduction, and there are

occasional VPCs (see Sections 6 and 8). The QRS complexes are extremely broad and bizarre because of two coexisting ECG abnormalities: right bundle branch block (RBBB) and WPW syndrome, type A (see Cases 19 and 195).

Case 207 Wolff-Parkinson-White Syndrome, Type A, Associated with Acute Anterior Myocardial Infarction and Left Ventricular Hypertrophy by Voltage

This ECG tracing was taken from a 54-year-old man with severe chest pain of 1 to 2 hours' duration. It shows sinus rhythm (rate: 60 beats/min) and WPW syndrome type A, associated with marked ST segment elevation and T wave inversion in leads V_1 through V_3 . This ST segment and T wave change is not due to WPW syndrome, and the ECG finding strongly indicates acute anterior myocardial ischemia or acute MI (see Cases 59 and 65). Later, acute MI was confirmed by other laboratory findings (markedly elevated serum enzymes) as well as clinical findings. The diagnosis of LVH is considered by voltage criteria alone (see Case 11).

Section 11

Uncommon Arrhythmias

Case 208 Ventricular Parasystole

The cardiac rhythm diagnosis is sinus rhythm (rate: 93 beats/min) with ventricular parasystole. The diagnosis of ventricular parasystole is made on the basis of varying coupling intervals with constant shortest interectopic intervals. Note a ventricular fusion beat (marked FB), which is commonly found in ventricular parasystole. (The numbers represent hundredths of a second.)

DIAGNOSTIC CRITERIA OF VENTRICULAR PARASYSTOLE

- Ventricular rhythm independent of the basic rhythm
- Varying coupling intervals
- Constant shortest interectopic intervals
- Long interectopic interval showing multiples of the shortest interectopic intervals
- Frequent appearance of ventricular fusion beats
- Usual rate ranging from 30 to 50 beats/min
- At times, a rate faster than the usual rates, resulting in parasystolic ventricular tachycardia

Regardless of its origin, parasystole is considered to be a benign and self-limited arrhythmia.

Case 209 Atrial Parasystole

Sinus rhythm with atrial parasystole (indicated by arrows). (The numbers represent hundredths of a second.) The diagnosis of atrial parasystole is made using the conventional criteria (see Case 208). The diagnostic criteria of atrial parasystole are essentially same as those of ventricular parasystole except that the origin of atrial parasystole is in the atria.

Case 210 Atrioventricular Junctional Parasystole

Sinus rhythm with atrioventricular (A-V) junctional parasystole (indicated by arrows). (The numbers represent hundredths of a second.) The diagnostic criteria of A-V junctional parasystole are essentially the same as those of ventricular parasystole (see Case 208), except that the origin of A-V junctional parasystole is in the A-V junction.

Case 211 Atrial Parasystole, Some Blocked

Arrows indicate ectopic P waves. The cardiac rhythm diagnosis is sinus rhythm (rate: 72 beats/min) with atrial parasystole (indicated by arrows), some blocked. Notice the varying coupling intervals with constant interectopic intervals, diagnostic of atrial parasystole (see Cases 208 and 209).

Case 212 Parasystolic Ventricular Tachycardia

This ECG tracing was discussed at the weekly ECG conference because of somewhat unusual cardiac arrhythmia. The cardiac rhythm diagnosis is a sinus rhythm (rate: 80 beats/min) with intermittent ventricular parasystolic tachycardia (rate: 72 beats/min), leading to incomplete A-V dissociation (see Cases 139 and 208). Arrows indicate sinus P waves. Note the frequent ventricular fusion beats of arying degree (marked X).

Case 213 Parasystolic Ventricular Tachycardia

A 22-year-old apparently healthy young man was referred to the cardiac clinic for the evaluation of his cardiac arrhythmia. The cardiac rhythm diagnosis is sinus arrhythmia (rate: 90–100 beats/min) and intermittent parasystolic ventricular tachycardia (rate: 95 beats/min), leading to incomplete A-V dissociation (see Cases 139 and 208). Note the frequent ventricular fusion beats (marked X). It has been shown that parasystolic ventricular tachycardia is a benign arrhythmia and is self-limited.

Case 214 Atrial Fibrillation with Intermittent Fascicular Parasystolic Tachycardia

The underlying cardiac rhythm is atrial fibrillation (AF) with advanced A-V block (ventricular rate: 76 beats/min). The arrhythmia diagnosis is intermit-

tent left posterior fascicular parasystolic tachycardia (rate: 72 beats/min) (see Case 208). The origin of the parasystole is considered to be in the left posterior fascicle of the left bundle branch system, in view of the incomplete right bundle branch block (RBBB) pattern with left anterior hemiblock (LAHB) pattern. In addition, hypokalemia should be considered because of the prominent U waves (leads V_4 through V_6) (see Case 10).

DIAGNOSTIC CRITERIA OF FASCICULAR RHYTHM OR TACHYCARDIA

- The QRS complexes of the ectopic rhythm or tachycardia show an incomplete RBBB pattern.
- Marked LAD (-45° or more) of the QRS complexes occurs when the ectopic impulses originate from the left posterior fascicle of the left bundle branch system.
- Marked RAD (+105° or more) of the QRS complexes occurs when the ectopic impulses originate from the left anterior fascicle of the left bundle branch system.
- The usual ventricular rate is 70 to 130 beats/min.

Case 215 Unilateral Slow Atrial Rhythm with Atrial Pacemaker Rhythm Producing Atrial Dissociation

The rhythm strips A to C are continuous. There are two independent atrial activities, neither disturbing the other; the ventricles are controlled by atrial pacemaker. Namely, this ECG rhythm strip reveals unilateral slow atrial rhythm (atrial rate: 96 beats) with an atrial pacemaker rhythm (rate: 101 beats/min), leading to atrial dissociation. Regularly occurring unilateral atrial P-P cycles can be appreciated by careful observation. Atrial dissociation superficially mimics atrial parasystole.

DIAGNOSTIC CRITERIA OF ATRIAL DISSOCIATION

- Atrial dissociation is an independent unilateral atrial rhythm localized in a portion of the atria; the unilateral atrial impulses are never conducted to the ventricles because of the intra-atrial block.
- Unilateral slow atrial rhythm is most common.
- Less common unilateral atrial rhythms may include atrial fibrillation, flutter, or tachycardia.

Case 216 Sinus Bradycardia with Unilateral Atrial Flutter Producing Atrial Dissociation

Leads II-a and II-b are not continuous. The underlying cardiac rhythm is marked sinus bradycardia (rate: 34 beats/min), but unilateral atrial flutter occurs intermittently, leading to atrial dissociation.

It should be noted that some QRS complexes are not preceded by P waves (e.g., the fifth beat in lead II-a and the second and fifth beats in lead II-b), meaning possible A-V JEBs. Atrial dissociation superficially resembles atrial parasystole, but unilateral atrial rhythm never activates the ventricles (see Case 215).

Case 217 Left Atrial Rhythm

The cardiac rhythm diagnosis is left atrial rhythm with a rate of 76 beats/min. Note that the ectopic P waves are inverted in leads I, II, III, aVF, and V₄ through V₆; lead V₁ exhibits only upright component of P wave. These unusual P waves are the characteristic features of left atrial rhythm.

DIAGNOSTIC CRITERIA OF LEFT ATRIAL RHYTHM

- Inverted P waves in leads II and aVF and the upright P wave in lead aVR (rarely biphasic or isoelectric P wave)
- Inverted P wave in lead I (at times, biphasic or isoelectric P wave)
- Lead V₁ with only a positive (upright) component of the P wave (a "dome and dart" P wave—a pathognomonic feature)
- Inverted P waves in leads V₄ through V₆
- PR interval of 0.12 to 0.20 second

Case 218 Wenckebach Sinoatrial Block Associated with Wenckebach Atrioventricular Block

This ECG tracing was taken on a 76-year-old man as an annual medical checkup. Arrows indicate P waves. The cardiac rhythm is sinus (atrial rate: 96 beats/min) with 5:4 Wenckebach A-V block associated with Wenckebach S-A block. Most readers should be able to recognize Wenckebach A-V block without any difficulty, but a coexisting S-A block is a rather difficult ECG abnormality to diagnose even for many cardiologists. For a better understanding of S-A block, every reader should study the Wenckebach A-V block in depth (see Cases 100 and 174). Note an A-V JEB (marked N) in rhythm strip lead II. Diaphragmatic MI is a remote possibility. Intermittent occurrence of longer P-P cycles is a key issue in identifying Wenckebach S-A block.

Case 219 Atrial Parasystole with Fascicular Escape Beats Causing Incomplete AV Dissociation Associated with Left Anterior Hemiblock and Probable Anteroseptal Myocardial Infarction

This ECG tracing, obtained from a 77-year-old woman, was presented to the weekly ECG confer-

ence because it reveals complex cardiac arrhythmias. The underlying cardiac rhythm is sinus (marked S), but there are regularly occurring ectopic P waves (indicated by arrows). This ectopic atrial rhythm represents atrial parasystole with a rate of 47 beats/min. Notice the varying coupling intervals with constant interectopic intervals—a characteristic feature of parasystole (see Cases 208 and 209). In addition, there are regularly occurring and slightly bizarre QRS complexes with incomplete RBBB pattern (marked X). These QRS complexes represent left posterior fascicular escape beats leading to incomplete A-V dissociation (see Case 214). Another ECG abnormality is LAHB (see Case 36). Furthermore, old anteroseptal MI should be considered.

Case 220 Atrial Flutter with 3:1 Atrioventricular Block Associated with Wolff-Parkinson-White Syndrome, Type A and Right Bundle Branch Block

A 71-year-old man was evaluated at the cardiac clinic for his frequent palpitations. The cardiac rhythm is atrial flutter (atrial rate: 204 beats/min) with 3:1 A-V block (ventricular rate: 68 beats/min) (see Section 6). The QRS complexes are extremely broad and bizarre because of two coexisting ECG abnormalities: RBBB and Wolff-Parkinson-White (WPW) syndrome, type A (see Cases 19 and 195). His atrial flutter is slower than usual because of the quinidine effect.

Case 221 Atrial Flutter-Fibrillation with Ventricular Parasystole and Probable Left Ventricular Hypertrophy

This ECG tracing was recorded from an 84-year-old man with chronic congestive heart failure (CHF). The underlying cardiac rhythm is atrial flutter-fibrillation (ventricular rate: 90–112 beats/min), but there are regularly occurring bizarre QRS complexes. Namely, there is ventricular parasystole (marked V) with a rate of 53 beats/min. Note the varying coupling intervals with constant interectopic intervals—characteristic feature of ventricular parasystole (see Case 208). Ventricular fusion beats (marked F) are also common in ventricular parasystole. LVH should be considered.

Case 222 Slow Ventricular Tachycardia

This ECG tracing was taken on a 74-year-old man with cardiomyopathy. The QRS complexes are

extremely broad and bizarre, and there is no discernible P wave. The cardiac cycle is precisely regular and all QRS complexes are upright (positive) in the entire precordial leads. Thus, the diagnosis is unequivocally slow ventricular tachycardia (VT) with a rate of 112 beats/min, because the broad QRS complexes are upright (positive) in the entire precordial leads (see Section 8 and Case 158). The only other diagnostic possibility is various supraventricular tachyarrhythmias associated with WPW syndrome, type A (see Case 195). Thus, these two diagnoses are the only possibilities whenever the broad QRS complex tachycardia reveals upright QRS complexes in all precordial leads (see Sections 8 and 10).

Case 223 Atrial Flutter with Wenckebach Atrioventricular Block Associated with Left Ventricular Hypertrophy

This ECG tracing, obtained from a 70-year-old woman with long-standing hypertension, shows a regular irregularity of the cardiac cycle in most areas. The cardiac rhythm is atrial flutter with Wenckebach A-V block; 2:1 and 4:1 A-V conduction ratios alternate, leading to a regular irregularity of the ventricular cycle in most areas. To understand Wenckebach A-V block during atrial flutter, every reader should study Wenckebach A-V block in depth during sinus rhythm (see Section 6 and Case 174). The diagnosis of LVH is obvious using the conventional criteria (see Case 11). In addition, old diaphragmatic MI is a remote possibility.

Case 224 Ventriculophasic Sinus Arrhythmia with Wenckebach Atrioventricular Block and Atrioventricular Junctional Escape Beats Producing Incomplete Atrioventricular Dissociation

An 82-year-old man was evaluated at the cardiac clinic because of a markedly slow heart rate. Arrows indicate sinus P waves. The underlying cardiac rhythm is sinus (atrial rate: 70 beats/min) with Wenckebach A-V block (best shown in the last section of the rhythm strip lead II) (see Case 174). In addition, there are frequent A-V JEBs (marked N) leading to incomplete A-V dissociation (see Case 139). The ventricular rate is about 40 beats/min. Another interesting ECG finding is ventriculophasic sinus arrhythmia; the P-P interval, including the QRS complex, is shorter than the P-P interval without QRS complex in most areas. Other than the cardiac rhythm abnormality, his ECG is within normal limits.

Case 225 Left Atrial Rhythm Associated with Wolff-Parkinson-White Syndrome, Type B, and Left Bundle Branch Block

This ECG tracing was taken on a 66-year-old man with extremely broad QRS complexes. Arrows indicate P waves. The cardiac rhythm is left atrial bradycardia with a rate of 57 beats/min. Note that the ectopic P waves are inverted in leads II, III, and aVF, and the P wave in lead V₁ exhibits only an upright (positive) component. This ECG finding indicates that the ectopic atrial impulse originates from the left atrium (see Case 217). The QRS complexes are extremely broad because of two coexisting ECG abnormalities: LBBB and WPW syndrome, type B (see Cases 26 and 194).

Case 226 Malfunctioning Pacemaker Manifested by Runaway Pacemaker and Pacemaker Electrode Malposition

A malfunctioning pacemaker (a fixed-rate unit) is manifested by pacemaker-induced ventricular tachycardia (rate: 167 beats/min; "runaway pacemaker"). In addition, the diagnosis of malposition of the pacemaker electrode is made on the basis of the upright QRS complex in lead V_1 . Remember that the QRS complex induced by artificial pacemaker should be negative in lead V_1 because the pacemaker electrode stimulates the right ventricular apex.

MALFUNCTIONS OF ARTIFICIAL CARDIAC PACING

- Acceleration of pacing (runaway pacemaker)
- Slowing of pacing
- Irregular pacing
- Failure of sensing
- Failure of capture
- Malposition of the electrode
- Any combination of the above

Case 227 Malfunctioning Pacemaker Manifested by Far-Advanced Runaway Pacemaker and Failure of Cardiac Capture

The pacing rate is extremely rapid (rate: 480 beats/min), so none of the pacing impulses are followed by the QRS complex. As a result, a preexisting complete A-V block has reappeared. This ECG tracing is an example of a far-advanced runaway pacemaker (see Case 226). Note a ventricular premature beat in lead V_1 .

Case 228 Malfunctioning Pacemaker Manifested by Far-Advanced Runaway Pacemaker and Failure of Cardiac Capture

Far-advanced runaway pacemaker is manifested by extremely rapid pacing (rate: 850 beats/min) with failure of ventricular capture (see Case 226). A pre-existing complete A-V block has reappeared (see Case 173). Diffuse myocardial ischemia and LVH should be considered (see Cases 11 and 55).

Case 229 Malfunctioning Pacemaker Manifested by Slowing of Artificial Pacing

The arrows indicate sinus P waves. A malfunctioning demand pacemaker is manifested by the extremely slow pacing rate of 31 beats/min (see Case 226). The preset pacing rate of this patient was 70 beats/min.

Case 230 Malfunctioning Pacemaker Manifested by Irregular and Slow Artificial Pacing

Extremely irregular and slow pacemaker-induced ventricular rhythm due to a far-advanced malfunctioning pacemaker (see Case 226). Note a long period of ventricular standstill in lead V₂.

Case 231 Artificial Pacemaker-Induced Ventricular Rhythm with Intermittent Atrial Capture

This recording exhibits an artificial pacemakerinduced ventricular rhythm with atrial captured beats occurring on every other beat (indicated by arrows). Note a long R-P interval.

Case 232 Artificial Pacemaker-Induced Ventricular Rhythm with Grouped Ventricular Premature Contractions and Ventricular Fibrillation Associated with Artificial Pacemaker Hysteresis

This recording exhibits an artificial pacemaker (demand unit)-induced ventricular rhythm with paroxysmal ventricular fibrillation because of the "R-on-T" phenomenon. The atrial mechanism is sinus. The R-R interval from the patient's natural beat to the next pacemaker beat is longer than the preset pacing interval because of *hysteresis*. The pacemaker functions normally.

Case 233 Artificial Pacemaker-Induced Ventricular Rhythm Associated with Ventricular Electrical Alternans

The cardiac rhythm is sinus, with an artificial pace-maker-induced ventricular rhythm (rate: 100 beats/min). It is interesting to note that the amplitude of the pacemaker beats alternate on every other beat. This unusual ECG finding is termed 2:1 ventricular electrical alternans involving the pacemaker-induced ventricular rhythm throughout (see Section 12).

Section 12

Miscellaneous ECG Abnormalities

Case 234 Mild Hyperkalemia

The cardiac rhythm is sinus tachycardia with a rate of 104 beats/min. Mild hyperkalemia is manifested by tent-shaped and peaking T waves with a narrow base involving many leads (see Case 49).

Case 235 Moderately Advanced Hyperkalemia

Tent-shaped and tall T waves with a narrow base are associated with slight nonspecific intraventricular block, and broad and low amplitude P waves are manifestations of moderately advanced hyper-kalemia (see Case 49). The ST segment elevation in leads V_1 through V_2 (pseudo anteroseptal myocardial infarction) is also caused by hyperkalemia. The cardiac rhythm is sinus with a rate of 70 beats/min.

Case 236 Far-Advanced Hyperkalemia Manifested by Bifascicular Block Consisting of Right Bundle Branch Block and Left Posterior Hemiblock

This ECG tracing was taken on a 48-year-old woman with chronic advanced renal failure. The cardiac rhythm is sinus tachycardia (rate: 110 beats/min) with first-degree A-V block. All P waves (indicated by arrows) are not easily visible because of their low amplitude. Far-advanced hyperkalemia is manifested by first-degree A-V block, flat P waves, bifascicular block (BFB) consisting of right bundle branch block (RBBB) and left posterior hemiblock (LPHB) and diffuse intraventricular block (see Cases 23 and 49). Pseudo diaphragmatic myocardial infarction (MI) is also produced by hyperkalemia.

Case 237 Far-Advanced Hyperkalemia Manifested by Diffuse (Nonspecific) Intraventricular Block

This tracing was obtained from a patient with refractory renal failure. The striking ECG abnormality is extremely broad QRS complexes caused by diffuse (nonspecific) intraventricular block as a result of faradvanced hyperkalemia (see Cases 48 and 49). In addition, pseudo diaphragmatic MI pattern is produced by hyperkalemia. The T waves are tall and the P wave amplitude is low.

Case 238 Mild Hypokalemia Associated with Left Atrial Enlargement and Diaphragmatic Myocardial Infarction

The cardiac rhythm is marked sinus bradycardia (rate: 50 beats/min) with first-degree A-V block. Note prominent U waves (marked U) diagnostic of hypokalemia (see Case 10). In addition, diaphragmatic MI and left atrial enlargement (LAE) are diagnosed using the conventional criteria (see Cases 6 and 65).

Case 239 Hypercalcemia and Left Ventricular Hypertrophy by Voltage Criteria

The cardiac rhythm is sinus tachycardia with a rate of 104 beats/min. The striking ECG abnormality is a markedly shortened QT interval because of virtual absence of the ST segment as a result of hypercalcemia. In addition, the diagnosis of left ventricular hypertrophy (LVH) is considered by voltage criteria alone (see Case 11).

HYPERCALCEMIA: ECG MANIFESTATIONS

- Earliest and most common ECG finding: Shortening of the QT interval due to the shortening or even absence of the ST segment
- Less common ECG findings: First-degree A-V block and diffuse intraventricular block
- Finding in advanced cases: Ventricular premature contractions (VPCs), ventricular tachycardia (VT), and ventricular fibrillation (VF)
- Additional findings: Synergetic action between digitalis and calcium

Case 240 Hypocalcemia Associated with Hyperkalemia

The rhythm is sinus with a rate of 95 beats/min. The QT interval is markedly prolonged primarily because of lengthening of the ST segment. This ECG finding is caused by hypocalcemia (see Case 50). Another electrolyte imbalance is mild hyper-kalemia manifested by peaked T waves with narrow base (see Case 49). In addition, diaphragmatic MI is a remote possibility.

Case 241 Advanced Hypocalcemia Associated with Hyperkalemia and Left Ventricular Hypertrophy

This ECG was obtained from a 37-year-old woman with severe renal failure. There are two major ECG

abnormalities due to combined electrolyte imbalances: advanced hypocalcemia (markedly prolonged QT interval as a result of a lengthening of the ST segment) and hyperkalemia (peaked T waves with narrow base) (see Cases 49 and 50). Another ECG abnormality is LVH (see Case 11).

Case 242 Acute Pericarditis Caused by Gunshot Wound

This ECG was obtained from a 23-year-old man with acute pericarditis as a result of a gunshot wound. The rhythm is marked sinus tachycardia with a rate of 138 beats/min. The ST segment elevation involving practically all leads is a characteristic feature of acute pericarditis.

PERICARDITIS: ECG MANIFESTATIONS

- Acute stage: Upward elevation of the concave ST segment involves diffusely every lead (except lead aVR).
- Subacute stage: The ST segment elevation returns to the isoelectric line, and the T waves begin to be inverted.
- Chronic stage: The T wave inversion may last for weeks or even months.
- Constrictive pericarditis or massive pericardial effusion: Low voltage of the QRS complexes are very common.

Case 243 Acute Pericarditis (Viral) and High Left Ventricular Voltage

This ECG tracing was taken on a 30-year-old man with acute pericarditis. The cardiac rhythm is sinus tachycardia with a rate of 120 beats/min. Acute pericarditis is manifested by the ST segment elevation involving practically every lead (see Case 242). In addition, high left ventricular voltage (HLVV) is present (normal variant) (see Case 3).

Case 244 Acute Pulmonary Embolism Manifested by Marked Sinus Tachycardia and P-Pulmonale

This ECG tracing was taken in the emergency room on a 57-year-old man with acute pulmonary embolism. Acute pulmonary embolism (PE) is manifested by marked sinus tachycardia (rate: 175 beats/min), atrial premature contractions (APCs; the second, fifth, and seventh beats in leads V₁ through V₃), P-pulmonale, and nonspecific ST segment depression in many leads.

ECG FINDINGS OF PULMONARY EMBOLISM

 Marked sinus tachycardia or various atrial tachyarrhythmias (e.g., atrial fibrillation, flutter, or tachycardia and multifocal atrial tachycardia)

- Right axis deviation of the QRS complexes
- RBBB
- P-pulmonale
- Inverted T waves in leads V₁ through V₃ (right ventricular strain pattern) and/or leads II, III, and aVF
- S_1Q_3 pattern or $S_1S_2S_3$ pattern (not common)

Case 245 Acute Pulmonary Embolism Manifested by Marked Sinus Tachycardia, Right Bundle Branch Block and Right Axis Deviation of the QRS Complexes

A 24-year-old woman was brought to the emergency room because of acute pulmonary embolism. The cardiac rhythm is marked sinus tachycardia with a rate of 152 beats/min. Acute pulmonary embolism is manifested by marked sinus tachycardia with RBBB of new onset and right axis deviation of QRS complexes (see Case 244).

Case 246 Acute Pulmonary Embolism Manifested by Marked Sinus Tachycardia, Right Bundle Branch Block, P-Pulmonale, and Right Axis Deviation of the QRS Complexes

This ECG tracing was taken on a 42-year-old woman with acute pulmonary embolism. The underlying cardiac rhythm is marked sinus tachycardia (rate: 148 beats/min), and there are occasional APCs (e.g., the second and third beats in leads V₁ through V₃). Acute pulmonary embolism is manifested by marked sinus tachycardia, APCs, RBBB, right axis deviation of the QRS complexes, P-pulmonale, and diffuse nonspecific ST segment depression (see Case 244).

Case 247 Central Nervous System Disorder Associated with Left Ventricular Hypertrophy and Anteroseptal Myocardial Infarction

The ECG abnormality in this patient is due to a central nervous system (CNS) disorder, a subarachnoid hemorrhage; obviously the markedly prolonged QT interval is a result of very broad and inverted T waves in many leads. Other ECG findings include LVH and anteroseptal MI (see Cases 11 and 65). All P waves are somewhat deformed and bizarre, but the cardiac rhythm is most likely sinus arrhythmia. Lead V_3 is half-standardized.

Case 248 Hypothermia Manifested by Sinus Bradycardia and Diffuse Intraventricular Block

This ECG tracing was obtained from a 21-year-old man who died following an automobile accident on an extremely cold day. He was found still alive in the snow after the accident. The cardiac rhythm is markedly slow sinus bradycardia (rate: 32 beats/min). The striking ECG abnormality is extremely broad and bizarre QRS complexes that represent typical ECG change because of hypothermia. Hypothermia resulted in the J-point ST segment elevation with notching of the terminal portion of QRS complex, termed an "Osborn wave" (see Case 51).

Case 249 Electrical Alternans and Probable Left Ventricular Hypertrophy

The cardiac rhythm is marked sinus tachycardia (rate: 152 beats/min), and the ECG diagnosis is 2:1 ventricular electrical alternans. Note that the QRS configuration alternates on every other beat.

In addition, the diagnosis of LVH should be considered (see Case 11).

DIAGNOSTIC CRITERIA OF ELECTRICAL ALTERNANS

- Ventricular electrical alternans: Alternating (commonly 2:1, rarely 3:1 or 4:1) QRS complexes, provided that every QRS complex originates from the same pacemaker.
- Atrial electrical alternans: Alternating P waves, provided that every P wave originates from the same pacemaker.
- Total electrical alternans: Combination of the ventricular and atrial electrical alternans.
- Repolarization electrical alternans: Alternating ST segments, T waves, and/or U waves.

Case 250 U Wave Electrical Alternans Associated with Right Bundle Branch

The cardiac rhythm is sinus with a rate of 63 beats/min. It is interesting to note that 2:1 electrical alternans involves only U waves (see Case 249). In addition, RBBB is diagnosed (see Case 19).

July Suggested Readings

Akhtar M. Cardiac arrhythmias and related syndromes: current diagnosis and management in cardiology clinics. Philadelphia: W.B. Saunders, 1993.

Allison T, Bardsley WT, Behrenbeck T, et al. Cardiovascular stress testing: a description of the various types of stress tests and indications for their use. Mayo Clin Proc 1996;71:43.

Bennett DH. Cardiac arrhythmias: practical notes on interpretation and treatment. 5th ed. Oxford: Butterworth-Heinemann, 1997.

Chakko S, Kessler KM. Recognition and management of cardiac arrhythmias. Curr Probl Cardiol 1995;20:53–120.

Chung EK. Principles of cardiac arrhythmias. 4th ed. Baltimore: Williams & Wilkins, 1989.

Chung EK. Pocket guide to ECG diagnosis. Malden, MA: Blackwell Science, 1996.

Chung EK. Pocket guide to exercise (stress) ECG testing. Malden, MA: Blackwell Science, 1997.

Chung EK. Manual of cardiac arrhythmias. Boston: Butterworth, 1986.

Chung EK. Electrocardiography: practical applications with vectorial principles. 3rd ed. Norwalk, CT: Appleton-Lange, 1985.

Chung EK. ECG diagnosis and self-assessment CD-ROM. Malden, MA: Blackwell Science, 1998.

Chung EK, Tighe DA. Pocket guide to cardiovascular diseases. Malden, MA: Blackwell Science, 1999.

Davis D. Differential diagnosis of arrhythmias. 2nd ed. Philadelphia: W.B. Saunders, 1997.

DiMarco JP, Prystowsky EN. Atrial arrhythmias: state of the art. New York: Futura, 1995.

El-Sherif N. Practical management of cardiac arrhythmias. New York: Futura, 1997.

Falk RH, Podrid PJ. Atrial fibrillation: mechanisms and management. New York: Raven Press, 1992.

Fisch C. Electrocardiography of arrhythmias. Philadelphia: Lea & Febiger, 1990.

Ganz LI, Friedman PL. Supraventricular tachycardia. N Engl J Med 1995;332: 162-173.

Isner JM. Right ventricular myocardial infarction. JAMA 1988;259:712.

Josephson ME. Clinical cardiac electrophysiology: techniques and interpretations. 2nd ed. Philadelphia: Lea & Febiger, 1993.

Klein LW, Helfant RH. The Q-wave and non-Q wave myocardial infarction: differences and similarities. Prog Cardiovasc Dis 1986;29:205.

Kulbertus HE, Rigo P, Legrand V. Right ventricular infarction: pathophysiology, diagnosis, clinical course and treatment. Mod Conc Cardiovas Dis 1985;54:1.

Marriott HJL. Advanced concepts in arrhythmias. 3rd ed. St. Louis: Mosby, 1998.

Marriott HJL. Advanced ECGs: a rapid review of diagnostic criteria. Philadelphia, PA: Trinity Press, 1997.

Naccarelli GV. Cardiac arrhythmias: a practical approach. New York: Futura, 1991.

Phibbs BP. Advanced ECG: board and beyond. Baltimore: Williams & Wilkins, 1997.

Podrid PJ, Kowey PR. Cardiac arrhythmias: mechanisms, diagnosis, and management. Baltimore: Williams & Wilkins, 1995.

Prystowky EN, Klein GJ. Cardiac arrhythmias: an integrated approach for clinician. New York: McGraw-Hill, 1994.

Roberts R. Recognition, pathogenesis, and management of non-Q wave infarction. Mod Conc Cardiovasc Dis 1987;56:17.

Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle branch block. N Engl J Med 1996;334:481.

Vlay SC. A practical approach to cardiac arrhythmias. 2nd ed. Baltimore: Williams & Wilkins, 1996.

Wagner GS. Mariott's practical electrocardiography. 9th ed. Baltimore: Williams & Wilkins, 1994.

Yamaki M, Ikeda K, Honma K, et al. Diagnosis of right ventricular involvement in chronic inferior myocardial infarction by means of body surface QRS changes. Circulation 1988;77:1283.

Zipes DP, Jalife J. Cardiac electrophysiology: from cell to bedside. Philadelphia: W.B. Saunders, 1995.

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ECG DIAGNOSIS:

A Self-Assessment Workbook

Edward K. Chung, M.D.

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- Each page presents a 12-lead ECG and/or ECG rhythm strips and an area for notes and suggested diagnosis
- Thorough analysis of ECG strips, rationale of diagnosis, and diagnostic criteria for abnormalities are provided by the author in the final section of the book
- This programmed text provides an easy way for students to learn this information in the absence of a formal course
- ECG Diagnosis: A Self-Assessment Workbook will be a companion volume to Pocket Guide to ECG Diagnosis and ECG Diagnosis and Self-Assessment CD-ROM by the same author

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